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Human body temperature is regulated by a proportionate control system. It is unclear, however, which internal body temperature is regulated by the control of the thermoregulatory effector responses of skin blood flow and sweating. The core temperature increases during exercise as a result of a "load error" and not a change in the regulated set-point temperature. During exercise the magnitude of core temperature elevation at steady-state is proportional to the metabolic rate and is largely independent of the environmental condition. However, dependent upon the environmental conditions the relative contributions of sensible (radiative and convective) and insensible (evaporative) heat exchange to the total heat loss will vary. The hotter the environment the greater the dependence on insensible heat loss. During exercise in the heat, the primary problem is to simultaneously provide the cardiovascular support to maintain the metabolism for skeletal muscle contraction and to dissipate the associated heat release. In hot environments, the core to skin temperature gradient is reduced to skin blood flow needs to be relatively high (compared to cooler environments) to achieve heat transfer sufficient for thermal balance. In addition, sweat secretion can

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Physiological Responses to Acute Exercise-Heat Stress

Michael N. Sawka, Ph.D. and C. Bruce Wenger, M.D., Ph.D.

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INTRODUCTION

This chapter discusses the normal physiological responses of humans performing exercise in the heat. Muscular exercise increases metabolic rate above resting levels, thus producing considerable heat release that needs to be dissipated to the environment in order to defend body temperature as well as protect the health of the individual. Compared with the broad temperature range for terrestrial climates (-88° to 58° C), the human body temperature must be regulated within a narrow range (24° to 43° C) to insure survival. To regulate body temperature, the human possesses an elaborate set of mechanisms, which include two distinct control systems: behavioral regulation and physiological regulation. During exercise in the heat, at least for highly motivated individuals, behavioral regulatory desires may be ignored. For physiological regulation the effector responses are: a) control of metabolic heat release; b) control of heat flow via the blood from the core to the skin, and c) control of sweating. During exercise without heat stress considerable cardiovascular support is required to support metabolism. When heat stress is combined with exercise, the cardiovascular system may be pushed to its limit to support the competing metabolic and thermoregulatory effector demands. This chapter will consider human temperature regulation, control of the thermoregulatory effector responses and the problems associated with maintaining adequate cardiovascular support during exercise-heat stress.

THERMOREGULATORY CONTROL

Behavioral Regulation

Selection of a micro-environment is the most primitive of the thermoregulatory responses, and is seen in all vertebrates, including fish and reptiles which, given the opportunity, will control their body temperatures by moving to a warmer or cooler

environment. By using clothing to change thermal insulation, and by using shelter, ventilation and air conditioning, human beings can live for extended periods in the most extreme climates on earth. Since behavioral thermoregulation involves conscious willed activity, it is more complex than physiological temperature regulation, and is less well characterized. Thermal sensation and thermal discomfort presumably represent the motivation for behavioral thermoregulation, and in human subjects, can be measured by psychophysical means. Although some workers (e.g., 30) have considered physiological and behavioral thermoregulation to be driven by the same error signal, it appears that during transients in environmental temperature, core and skin temperatures change much more slowly than do thermal sensation and thermal discomfort (61). Since changes in physiological thermoregulatory responses are nearly proportional to changes in core and skin temperatures, it would seem that they will trail behind changes in sensation and discomfort. Thermal sensation and discomfort thus seem to have an anticipatory component (perhaps related to rate of change of skin temperature), which may have some value teleologically, since behavioral thermoregulation is accomplished by discrete responses, whereas physiological thermoregulation is accomplished by responses which occur continuously, and whose intensity may be adjusted, as long as the thermal stress is present.

Among the most familiar examples of non-living regulatory systems are ovens and heating systems for buildings, which are controlled by thermostats which operate so as to maintain the temperature inside the oven or building at a relatively constant level. Typically, such a heating system controlled by a thermostat can operate at only two levels of heat production, on and off, and is not capable of a graded response. When the temperature inside the oven or building falls below the desired level, the thermostat turns on the heat source (furnace, boiler, or heating coil); and when the temperature rises above that level, the thermostat turns off the heat source. In practice the

response of such a system lags somewhat, so that it is not constantly switching on or off in response to small temperature changes. Such a system is called an on-off system. The quantity which a control system acts to maintain within narrow limits (in this example, temperature) is often called the regulated variable, and the quantities which it controls in order to accomplish this (in this example, rate of heat production averaged over several on-off cycles) are called controlled variables.

Physiological Regulation

Rather than operating only at one of two levels, on and off, most physiological control systems can produce a graded response according to the disturbance in the regulated variable. In many instances, changes in the controlled variables are proportional to displacements of the regulated variable from some threshold value, and such control systems are called proportional control systems. Figure 3-13 shows the dependence of sweating, skin blood flow, and capacity of superficial veins on core and mean skin temperatures. Note that the temperature controller is of the proportional type, and that these thermoregulatory effector responses depend not only on core temperature, but also on the mean skin temperature. Although the most accurate mathematical representation of the control of the thermoregulatory effectors may well be a complicated function of tissue temperatures at a number of sites, and contain non-linear elements, it is convenient and useful to represent it in terms of a linear combination of core temperature (T_c) and mean skin temperature (T_{sk}), and to represent the control of each thermoregulatory effector response by an equation of the form:

$$R - R_o = \underline{a}_1 \cdot T_c + \underline{a}_2 \cdot T_{sk} + \underline{b} \qquad (1)$$

In this equation R is a thermoregulatory response, R_o is a basal value of R, and $\underline{a}_1$, $\underline{a}_2$, and $\underline{b}$ are constants. In terms of Figure 3-13, $\underline{a}_1$ is the slope of a particular R:T_c relation and $\underline{a}_2$ is the effect of a unit change in T_{sk} on the R:T_c relation. The ratio $\underline{a}_1$:$\underline{a}_2$ is about 9:1 for all of the heat dissipating responses: sweating (92), vasodilation as represented by forearm blood flow (165), and vasoconstriction as represented by finger blood flow (164).

The sensitivity of these thermoregulatory effector responses to mean skin temperature allows the thermoregulatory system to respond appropriately to changes in environmental temperature over a wide range with little change in body core temperature. For example, someone who enters a hot environment will begin sweating because of an elevated skin temperature before any possible change in core temperature. On the other hand, an increase in heat production within the body, as occurs during exercise, has relatively little effect on skin temperature and thereby elicits the appropriate heat-dissipating responses, mostly through the rise in core temperature. Once these thermoregulatory effector responses are sufficient to dissipate heat at the rate to which it is being produced, thermal balance is restored and core temperature achieves steady-state levels. In the language of control theory, the rise in core temperature which elicits the heat-dissipating responses necessary to re-establish thermal balance is called a "load error." Even in a cold environment, core temperature must rise during exercise in order to elicit vasodilation to transfer heat to the skin, whether or not sweating also is required. In such a case, the vasodilation occurring at a given level of core temperature will be less than in a warm environment, because of the lower mean skin temperature; but the lower mean skin temperature will also allow the necessary core-to-skin heat transfer to be achieved with less skin blood flow than in a warm environment.

Core temperature during exercise increases in proportion to the metabolic rate regardless of the environmental temperature over a fairly wide range. This was first observed experimentally in 1938 by Marius Nielsen (103), who showed that during 1 hours' exercise in ambient temperatures from 10 to 35° C, changes in heat loss by conduction and convection (dry or "sensible" heat loss) were exactly balanced by changes in evaporative cooling ("insensible" heat loss). Nielsen concluded that the rise in core temperature during exercise was the consequence not of an insufficient ability to dissipate heat, as had previously been thought, but of "a change in the regulatory adjustment of body temperature" (104). Although he did not specify how he conceived such a change to affect the control of thermoregulatory effector responses, his conclusion has often been understood in such a way as to indicate an elevation of the threshold levels of core temperature for the thermoregulatory effector responses such as occurs, for example, in fever (51,90). We have already accounted for the rise in core temperature as a load error, without invoking any such threshold changes. We will comment further on the difference between the rise in core temperature with exercise and that with fever after developing the concept of thermoregulatory "set point".

Besides fever, a number of other factors, such as heat acclimation (Chapter 4), and phase of the menstrual cycle and time of day (Chapter 7), change the level at which the thermoregulatory system operates. Since some of these factors have been shown to shift thresholds for several different thermoregulatory effector responses by the same amount (see References 54,70 for further discussion) it is useful to speak of a thermoregulatory "set point", which serves as a reference temperature in the control of the effector responses, and which is changed by these factors. Figure 3-1 shows schematically the difference between the rise in core temperature with exercise and that with fever. Conceptually, the difference between fever and exercise in Figure 3-1 is that the former involves an increase in thermoregulatory set point, and the latter does

not (143). The observations on which this conceptual difference is based are these: First, if substantially increased heat production occurs at all during fever, it occurs only during the phase of rising core temperature, but is not necessary to maintain the fever; whereas during exercise, increased heat production not only causes the elevation in core temperature, but is necessary to sustain it. Second, the heat dissipating responses are lower than their pre-febrile levels for almost the whole time that core temperature is rising during fever, whereas during exercise the heat dissipating responses start to increase early, and continue increasing with rising core temperature.

[INSERT FIGURE 3-1 HERE]

The function of the human thermoregulatory system is shown schematically in Figure 3-2. Certain factors that act peripherally on the sweat glands and blood vessels, and are associated with local temperature and heat acclimation, are omitted here, but will be discussed later in this chapter and in Chapter 4. The scheme presented in Figure 3-2 presumes that there are temperature receptors in the skin and body core which send information about their temperatures to some central integrator, which generates a "thermal command signal" that participates in the control of sweating, vasodilation, and vasoconstriction. The notion of such a thermal command signal is supported by the evidence for a common thermoregulatory set point, mentioned above, and by evidence that core and skin temperature are combined in the same way in the control of sweating, vasodilation, and vasoconstriction (54). Division of thermosensitivity in the body into skin and core is convenient and useful in practice, but it is somewhat artificial since, as discussed later in the chapter neither the skin nor the core have a uniform temperature most of the time. The core thermosensitive structures (or at least those that participate in the control of thermoregulatory

responses) are very unevenly distributed, and in laboratory mammals are concentrated especially in the hypothalamus, which also is where much of the integration occurs (14). Changes of only a few tenths of a degree in the temperature of the anterior preoptic area of the hypothalamus can elicit thermoregulatory effector responses, and this area contains many neurons which increase their firing rate in response either to heating or to cooling. A large share of the central temperature receptors in human beings is presumed to be in the hypothalamus, although there is no direct evidence on this point. There is evidence, however, for a high concentration of thermosensitive structures in the distribution of the carotid artery, since infusions of hot saline solution into the carotid artery cause more vasodilation in the hand than do infusions into other arteries (31). It is not known how much temperature sensors inside the core but outside the hypothalamus contribute to thermoregulation, but central temperature receptors have been reported also in the heart of rabbits (39), pulmonary vessels of sheep (13), and spinal cord of dogs (153).

[INSERT FIGURE 3-2 HERE]

Since skin temperature generally is not uniform, the contribution of skin temperature to thermoregulatory control is usually expressed in terms of an appropriately weighted mean skin temperature. Neurophysiological studies show three types of thermal receptors in skin: One responds to heating with a transient burst of activity and an increase of static activity; one responds to cooling with a transient burst of activity and an increase of static activity (60,64); and one responds to several stimuli, including warming, with a continuous discharge (60). The transient discharges that occur at the beginning of heating or cooling provide the central integrator with information about changes in skin temperature as they are occurring, and this feature

may account for the sensitivity of sweating not only to skin temperature, but also to the rate at which skin temperature is changing (92).

BODY TEMPERATURES

Core Temperature

Fundamental to the study of human temperature regulation is the quantification of deep body or core temperature. Core temperature, is measured during physical exercise, to either estimate average internal temperature or provide information concerning the contribution of core temperature to the control of thermoregulatory effector responses. It might seem that this basic measurement should by now, more than a century since the pioneering work of Claude Bernard (9), be well defined and broadly accepted. Instead, there is still debate among thermal physiologists concerning the best measurement site and the correct interpretation of the different indices of core temperature (15).

The idea that a single temperature measurement can provide an index of the average internal temperature may not be reasonable. By 1950, it was clear to several investigators that the notion of a single measurement site representing the average internal temperature was fictitious (7,8,66,85). In fact, Mead and Bonmarito (85) stated that "it is apparent that no single regional temperature could indicate, except by chance, the average of all internal temperatures". The idea that a single temperature measurement can provide a close approximation of the regulated temperature has several problems, both theoretical and practical. First, it is not known what single temperature or integrated sum of core temperatures are being regulated (54). Second, although the hypothalamic temperature is believed to be a major input into the

regulated temperature, neither brain temperature (167) nor hypothalamic temperature has been well quantitated in humans, nor will they be measured in human subjects until non-invasive measurement techniques become available.

The temperature within a given body region is dependent upon: (a) the metabolic rate of the surrounding tissues; (b) the source and magnitude of blood flow; and (c) the temperature gradients between contiguous body regions. Considerable temperature gradients exist between and within different orifices, body cavities and blood vessels (43,66,86,141). For resting humans, about 70% of the metabolic heat is produced by internal organs and viscera within the body core (3,145). During muscular exercise, however, about 90% of the metabolic heat can be produced by the skeletal muscles. Because of the different source of metabolic heat during exercise as compared to rest, temperatures measured during exercise within a given body region may change relative to other body regions (2,86,123,124). For example, during rest in a comfortable environment, skeletal muscle temperature is lower than core temperature; however during exercise, the temperature within the active skeletal muscle exceeds core temperature (2,123,124), while the temperature within the inactive skeletal muscles usually does not increase (2). Blood perfusing the active skeletal muscles is warmed, and the warmer blood carries heat to other body regions, and consequently core temperature is elevated (2,86).

The general requirements for an ideal site to measure core temperature are: (a) the measurement site should be convenient; (b) the measurement should not be biased by environmental conditions; and (c) the measured changes should quantitatively reflect small changes in arterial blood temperature (28,141). In addition, the response time for temperature changes is an important consideration when selecting a measurement site for certain experimental approaches. For experiments employing exercise in the heat, the subject's core temperature can be measured at the esophagus, rectum, mouth

and tympanum/auditory meatus. The relative advantages and disadvantages of these and other measurement sites are well understood (2,29,32,52,81,124). During the subsequent paragraphs these core temperature measurement sites will be systematically evaluated for their compliance with the stated requirements for an ideal site as well as for their correct usage.

Esophageal Temperature

Esophageal temperature is obtained by inserting a catheter (containing a thermocouple or thermistor) through the nasal passage and into the throat and then swallowing it. Since some individuals find this procedure to be uncomfortable, a topical anesthetic, such as lidocaine, is often used on the catheter and/or in the throat to alleviate some discomfort. It is important to use a fairly stiff plastic catheter which will not easily curl when the probe is advanced within the esophagus. The catheter is secured (by taping to the dorsum of the nose) once the temperature sensor is at the level of the left atrium. At approximately this level, the heart and esophagus are in contact and are fairly isothermal (15). If the catheter is passed too far, such as into the stomach, the temperature and response times are altered; likewise, if the catheter is too high, the pulmonary ventilation will cool or heat (dependent upon ambient conditions) the temperature sensor (15,32,68,168). The proper catheter length can be predetermined from anatomical measurements and may be verified by a radiogram, an acoustical signal, or by monitoring an electrocardiogram from the catheter tip (17). In our laboratory, an anatomical measurement of one-fourth the subject's stature (R.R. Gonzalez, unpublished anatomical measurements of cadavers) is used as the catheter distance from the nostril to the heart level within the esophagus. A final methodological consideration is the avoidance of swallowing during measurement, as the

passage of saliva will spuriously lower the esophageal temperature values. Swallowing can be avoided by having the subject spit or by using a suction device to remove saliva from the mouth (15).

Most thermal physiologists agree that esophageal measurements provide the best non-invasive index of core temperature for humans. Several investigators have found a rapid response of esophageal temperature to changes in blood temperature elicited by extracorporeal circulation (89,141). and by body cooling during anesthesia (29). For example. Shiraki et al. (141) found that esophageal temperature quantitatively reflected pulmonary artery (mixed venous blood) temperature with an average difference of -0.1° C and a lag time of about one minute during their hyperthermia experiments. Therefore, esophageal temperature provides a good index of blood temperature. Several investigators have simultaneously measured esophageal temperature and the temperature of active skeletal muscles during exercise (2,122,123,124). Each study demonstrated that during exercise esophageal temperature and active muscle temperatures achieved steady-state values in 15-20 minutes. The rapid response time for esophageal temperature is due to the low heat capacity of the esophagus and its proximity to the heart; therefore, it can be rapidly warmed or cooled.

Rectal Temperature

Rectal temperature is widely employed by physiologists because it is the most comfortable and non-invasive measurement site. Rectal temperature is obtained by inserting a temperature sensor a minimum of five centimeters past the anal sphincter. Temperature measurements are uniform within the rectum because there is no thermal gradient from 5 to 27 centimeters past the anal sphincter (1,8,71,102). One early study (85) reported that rectal temperature values varied by as much as 0.8° C

depending upon the insertion depth; this variability apparently resulted from the use of a very flexible catheter which caused great variability for temperature sensor placement within the pelvis. The use of a more rigid rectal catheter seems to solve this problem (1,8,71,102). Another potential problem is that a properly placed temperature sensor may slip to less than five centimeters beyond the anal sphincter during the performance of muscular exercise. This potential problem can be solved by attaching a bulb to the rectal catheter, at the desired insertion depth, that will abut the anal sphincter.

Rectal temperature values are generally higher than values measured in arterial blood (6,43) and other core temperature sites. During exercise it takes approximately 25-40 minutes to achieve steady-state rectal temperature values (2,58,85,102,122,123). These steady-state values are generally ~0.2° C higher than simultaneously measured esophageal temperature values (2,102,122,123) and they are independent of the environmental temperature (57,95,146). As a result, the steady-state rectal temperature provides a good index to assess body heat storage (124,148). The main problem with rectal temperature is that it is slow to respond to changes in blood (86) and other core temperatures. Figure 3-3 provides a representative record of rectal and esophageal temperature responses to two exercise bouts spaced by a rest period. Note that the slow response results in rectal temperature being less than esophageal values during periods of rapid heat storage and greater than esophageal values during periods of rapid heat loss. The reason for the slow response of rectal temperature to thermal transients is probably a low rate of blood flow to the rectum compared to other measurement sites (6,89). The slow response time makes rectal temperature a poor core temperature index for estimating the drive for effector responses (42,52,123).

[INSERT FIGURE 3-3 HERE]

Oral Temperature

Oral temperature measurements are routinely made by physicians to determine the presence of fever. For this, a thermometer is placed sublingually and allowed to equilibrate prior to recording of the temperature. The equilibration time necessary to obtain a stable oral temperature value ranges from 3 to 10 minutes and is dependent upon the ambient temperature (81,146). During the equilibration and measurement period the subject must breathe through the nose, as ventilation from mouth breathing can artificially lower oral temperature values in cold environments and artificially elevate oral temperature values in hot environments. Some investigators have sealed their subjects' mouths to insure nasal breathing while obtaining oral temperature measurements (32,52). Finally, both swallowing and the ingestion of fluid must be avoided as they will bias oral temperature values (32,148).

The tongue is perfused by the lingual branch of the external carotid artery and has a high blood flow per gram of tissue (63). This enables the tongue to be an effective heat exchanger and allows sublingual temperatures to approximate blood temperature (67); however, changes in head and face skin temperatures will bias oral temperature (84,142). McCaffrey et al. (84) measured oral temperature at both sides of the mouth while simultaneously cooling and heating contralateral areas of the head and neck. The oral temperature values followed the changes in cutaneous temperature that occurred on the same side of the head. Also, in cold environments, cheek cooling will affect the superficial parotid saliva duct and cool the saliva reaching the mouth, thus biasing the temperature measurement (142). Therefore, unlike esophageal and rectal temperature values, oral temperature values are not always independent of the environmental temperature.

Under ideal conditions, oral temperature values are very similar to esophageal temperature values (32,41,52,81). During steady-state light intensity exercise, which can be performed with nasal breathing, oral temperature values have been found to correlate well (r=0.83-0.92) with simultaneously measured esophageal and rectal temperature measurements (81,147). During steady-state moderate intensity exercise, which can be performed with nasal breathing, oral temperature values may be lower than esophageal and rectal temperature values, perhaps due to a sympathetically mediated vasoconstriction of the tongue vasculature (81,148). Another possibility, is that additional cooling of the upper respiratory passages may occur at greater exercise intensities (148).

Tympanic/Auditory Meatus Temperature

Tympanic temperature is obtained by inserting a small temperature sensor into the ear canal and advancing it until it rests against the tympanic membrane. Proper placement is determined by the subject's hearing a sound when the temperature sensor touches the tympanic membrane. Some subjects find this contact to be uncomfortable (15). In addition, there are reports of the temperature sensor's perforating the tympanic membrane (36,149,156). Because of the potential discomfort and trauma as well as placement problems associated with tympanic measurements, some investigators have chosen instead to measure the temperature of the external auditory meatus. For this measurement, a temperature sensor is placed in an ear plug and inserted into the external auditory meatus. Placement of the temperature sensor is important since there is a substantial (~0.5° C) temperature gradient along the wall of the meatus (28).

Tympanic/auditory meatus temperature measurements do not provide a reliable index of the level of core temperature during either rest or exercise (28,57,82,83,84,95).

Depending upon the environmental conditions, tympanic/auditory meatus temperature values can be lower or higher than simultaneously measured steady-state rectal (57,95) and esophageal (82,83,84) temperature values. In addition, local head heating and air flow to the face will bias the temperature of the external meatus (82,83,84). Therefore, tympanic/auditory meatus temperature is not independent of the environmental conditions. Since the tympanic/auditory meatus temperature values are biased by facial skin temperature, the measured temperature is the sum of a variable weighting (depending upon the environmental condition) of skin and core temperature (57,85,95). However, if ambient temperature is constant and neither esophageal or oral measurements can be obtained, the change in tympanic/auditory meatus temperature, because of its rapidity, can be of some value during exercise (109).

Skin Temperature

Skin temperature is measured for the purposes of: (a) calculating the mean body temperature for heat storage determinations; (b) calculating sensible (radiative and convective) heat exchange and skin conductance; and (c) integrating into an index of the thermal drive for the thermoregulatory effector responses. Although the skin surface is easily accessed (unlike the core), measurement problems often occur because the skin represents the boundary between two media, tissue and the ambient air. As a result, changes in skin temperature might result from physiological adjustments (cutaneous blood flow, sweat evaporation) or alterations in the ambient environment (air motion, temperature, radiation). Generally, skin temperatures are measured from temperature sensors in contact with the skin's surface or from spectral methods. For the former method, care needs to be taken to insure that the temperature sensor remains in good thermal contact with the skin, since otherwise the measurement will be biased by the ambient temperature.

Although a single skin temperature measurement can be useful for biophysical calculations, thermal physiologists are more often interested in the average or mean skin temperature (62,170). The mean skin temperature (T_{sk}) represents the sum of weighted individual skin temperatures. Generally, the weighting is based on the percentage of body surface area that is represented by the body region from where the temperature is measured (172). For example, Hardy and DuBois (62) divided the body into twelve regions while Winslow and colleagues (171) divided the body into fifteen regions for skin temperature measurements. Numerous investigators have attempted to minimize the number of measurement sites necessary to obtain a valid estimate of mean skin temperature (110,151). Mitchell and Wyndham (87) compared nine of these shortened equations which are used to estimate mean skin temperature. They recommended that when it is difficult to measure a large number of sites, the equation developed by Ramanthan (110) be used, where:

$$T_{sk} = 0.3\ (\text{chest+upper arm temperatures}) + 0.2\ (\text{thigh + calf temperatures}) \quad (2)$$

Mean skin temperature values that are based on regional weighting according to the percentage of body surface area are particularly useful for the calculation of mean body temperature and heat storage.

Nadel and colleagues (97) have developed a novel approach to determine the regional weightings for the mean skin temperature calculation. Their approach was to base the regional weighting on the skin's thermal sensitivity and not on its percentage of body surface area. These investigators reasoned that thermal receptors are not evenly distributed over the skin's surface, and warming of a body region having the greatest number of thermal sensors would have the greatest influence on the thermoregulatory effector responses. Nadel and colleagues had subjects rest in a range from warm to hot environments while they applied thermal radiation to selected skin

areas and measured the change in sweating rate at the thigh. Based upon the thermal sensitivity data (change in thigh sweating rate) which was adjusted for regional surface area, they developed the equation, where:

T_{sk} = 0.21 (face temperature) + 0.21 (chest and back temperatures) + 0.17 (abdomen temperature) + 0.15 (thigh temperature) + 0.08 (calf temperature) + 0.12 (upper arm temperature) + 0.06 (forearm temperature) (3)

Subsequently, Libert et al. (77) conducted a similar study in which they heated larger skin areas by smaller temperature increments but for greater durations than in the experiments of Nadel and colleagues. Libert et al. (77) concluded that the weighting factors based on thermal sensitivity were not different from the weightings based on percent of body surface area. It seems that more research is needed concerning the use of a thermal sensitivity approach for calculation of the mean skin temperature. Both of these studies were performed on semi-nude and resting subjects (77,97), and the results obtained may not be directly applicable to clothed or exercising subjects.

CORE TEMPERATURE RESPONSES TO EXERCISE

Environmental and Exercise Intensity

As depicted in Figure 3-3, during dynamic exercise core temperature initially increases rapidly and subsequently increases at a reduced rate until essentially steady-state values are achieved. The elevation of core temperature represents the storage of metabolic heat which is released as a by-product of skeletal muscle contraction. During the initiation of exercise, the metabolic rate increases immediately; however, the thermoregulatory responses for heat dissipation respond more slowly. The

thermoregulatory effector responses which enable sensible (radiative and convective) and insensible (evaporative) heat loss to occur, increase in proportion to the metabolic heat release (99). Eventually, these heat loss mechanisms increase sufficiently to balance metabolic heat, allowing a steady-state core temperature to be achieved.

During muscular exercise, the magnitude of core temperature elevation at steady-state is largely independent of the environmental condition and is proportional to the metabolic rate (78,103,104,146). These concepts were first presented by Nielsen (103) who had three subjects perform exercise at several intensities (up to ~3.0 $\ell O_2 \cdot min^{-1}$) in a broad range (10° to 35° C with low humidity) of ambient temperatures. Figure 3-4 presents the heat exchange data for one subject during 1-h cycle exercise at an aerobic metabolic rate of approximately 650 watts. The difference between metabolic rate and total heat loss represents the energy used for muscular contraction and the heat storage. Note that the total heat loss and, therefore, the heat storage and elevation of core temperature were constant for each environment. The relative contributions of sensible and insensible heat exchange to the total heat loss, however, varied with the environmental conditions. In the 10° C environment, the large skin-to-ambient temperature gradient facilitated sensible heat exchange which accounted for ~70% of the total heat loss. As the ambient temperature increased, this gradient for sensible heat exchange diminished and there was a greater reliance upon insensible heat exchange. When the ambient temperature is equal to skin temperature, insensible heat exchange will account for essentially all of the heat loss. In addition, when the ambient temperature exceeds the skin temperature, there is a sensible heat gain to the body.

[INSERT FIGURE 3-4 HERE]

Nielsen's finding that the magnitude of core temperature elevation is independent of the environmental conditions is inconsistent with the personal experience of most athletes. For example, a runner will certainly experience greater hyperthermia if he/she competes in a 40° C than in a 20° C environment (113). Lind (78) has shown that the magnitude of core temperature elevation during exercise is independent of the environment only within a range of conditions or a "prescriptive zone". Figure 3-5 presents a subject's steady-state core temperature responses during exercise performed at three metabolic intensities while in a broad range of environmental conditions. The environmental conditions are represented by the "old" effective temperature, which is an index that combines the effects of dry bulb temperature, humidity and air motion. Note that during exercise, the greater the metabolic rate, the lower is the upper limit of the prescriptive zone. In addition, Lind found that even within the prescriptive zone there was a small but significant positive relationship between the steady-state core temperature and the old effective temperature. It seems fair to conclude that throughout a wide range of environmental conditions, the magnitude of core temperature elevation during exercise is largely but not exclusively independent of the environment. During exercise with a substantial combined metabolic and environmental heat stress, the thermoregulatory load error is great enough to result in additional steady-state core temperature levels.

[INSERT FIGURE 3-5 HERE]

As stated, within the prescriptive zone the magnitude of core temperature elevation during exercise is proportional to the metabolic rate (103,124,146). The greater the absolute metabolic rate, the higher the individual's steady-state core temperature during exercise. Saltin et al. (123) reported that within the prescriptive

zone, the relationship of steady-state core temperature to metabolic rate during exercise can be described by the following equation:

$$T_c = 0.00408\ M + 35.9 \quad (r=0.83,\ n=3)$$

Where: T_c is the steady-state core temperature in °C, and

M is the absolute metabolic rate in Watts per square meter

The relationship between absolute metabolic rate and core temperature is good for a given individual, but does not always hold well for comparisons between different individuals. Åstrand (5) first reported that the use of relative intensity (percent of maximal oxygen uptake), rather than absolute intensity, removes most of the inter-subject variability for the core temperature elevation during exercise. Figure 3-6 shows these relationships for subjects who performed exercise at absolute and relative intensities in a moderate environment (124).

[INSERT FIGURE 3-6 HERE]

Davies and colleagues (33,34,35) attempted to clarify the relationship between relative intensity and the steady-state core temperature response to exercise. Their investigations were performed in environments with dry bulb temperatures ranging from 5° to 25° C (relative humidity is <50%) and with the subjects performing exercise at relative intensities ranging from ~20% to ~90% of their maximal oxygen uptake. Figure 3-7 presents data redrawn from their studies: note that for a group of laboratory and field experiments (Figure 3-7A) there was a curvilinear relationship between steady-state core temperature and relative intensity (35), described by the quadratic equation:

$$T_c = 37.25 - 0.00264\ (\%\dot{V}O_2max) + 0.00037\ (\%\dot{V}O_2max)^2$$

Figure 3-7B presents two subjects' steady-state core temperature values during exercise, at 65% and 85% of their maximal oxygen uptake, in relation to the ambient dry bulb

temperature (33). Core temperature was found to be independent of dry bulb temperature at the 65% relative intensity: however, core temperature was influenced by dry bulb temperature at the 85% relative intensity. These data are somewhat consistent with those of Lind (78) showing that the prescriptive zone is smaller at higher metabolic rates.

[INSERT FIGURE 3-7 HERE]

The preceding investigations have shown that in humans, core temperature changes are related to the relative exercise intensity. It would, therefore, seem logical to expect that any condition lowering maximal oxygen uptake (and thus increasing relative intensity) would also elicit an elevated core temperature response at a given absolute (thus higher relative) intensity. This hypothesis has been tested by several investigators with differing results (58,74,101,121). Greenleaf et al. (58) lowered two subjects' maximal oxygen uptake by simulating altitudes of 2,000 m and 3,000 m in a hypobaric chamber. Their subjects' core temperature responses during cycle exercise at a given absolute intensity were similar whether exposed to sea level or to simulated altitude. Rowell et al. (121) lowered six subjects' maximal oxygen uptake by having them breathe hypoxic (11-12% O_2) gases during cycle exercise in a comfortable environment. They found similar core temperature responses during exercise at a given absolute intensity whether the subjects were breathing normoxic or hypoxic air. In contrast, Nielsen (101) found that lowering of maximal oxygen uptake by carbon monoxide poisoning resulted in a higher core temperature during cycle exercise at a given absolute intensity, but the relation to relative intensity was essentially unchanged. Finally, Kolka et al. (74) had eight subjects perform cycle exercise in a hypobaric chamber at sea level, and then at simulated altitudes of 2,600m and 4,600m. They

found that the change in core temperature during exercise was dependent upon the relative intensity.

It would also seem logical to expect that any condition increasing maximal oxygen uptake (thus lowering relative intensity) would elicit a lowered core temperature at a given absolute intensity. Two studies have used autologous erythrocyte reinfusion to increase subjects' maximal oxygen uptake (comfortable environment) and subsequently measured their core temperature responses to exercise in the heat (132,133). The first study (132) used subjects who were unacclimated to the heat, and found that steady-state core temperatures at a given absolute intensity were not significantly reduced after erythrocyte reinfusion. However, the calculated heat storage values tended to be lower during the post- than during the pre-reinfusion heat stress tests. The second study (132) used heat-acclimated subjects, and found lowered core temperature responses at a given absolute intensity during the post- compared to the pre-reinfusion heat stress tests.

In conclusion, during muscular exercise the core temperature response is proportional to the metabolic intensity. If the metabolic intensity is expressed as a percentage of the individual's maximal oxygen uptake, then much of the inter-subject variability disappears for the magnitude of steady-state core temperature values. Acute changes in maximal oxygen uptake, however, may or may not modify the steady-state core temperature response at an absolute metabolic intensity. These inconsistent results probably reflect differences in subject status (e.g., fitness, acclimation state) and/or differences in the methods employed to acutely alter maximal oxygen uptake.

Exercise Type

All of the investigations discussed so far have employed leg (lower body) exercise (treadmill or cycle). There is debate as to whether upper body exercise (e.g., arm-crank) results in different core temperature values than those elicited by lower body exercise at the same absolute metabolic rate. Maximal effort arm-crank exercise elicits an oxygen uptake that is approximately 70% of that obtained during maximal effort cycle exercise. It is uncertain whether the core temperature response during arm-crank exercise would be coupled to the relative intensity of the upper or lower body muscle mass. If these responses are coupled to the peak oxygen uptake of the musculature employed, then arm-crank exercise would be expected to elicit a higher core temperature for a given absolute intensity than would lower body exercise.

In 1947, Asmussen and Nielsen (4) studied two subjects' core temperature responses to arm-crank and cycle exercise at equivalent metabolic rates. They found that arm-crank exercise elicited lower rectal temperature values than cycle exercise. In addition, this difference widened as metabolic intensity increased. Since the authors were concerned that rectal temperature may have been spuriously high during cycle exercise because of the warm venous blood returning from the leg muscles, they conducted additional experiments in which they monitored stomach temperature. In agreement with their rectal temperature data, the stomach temperature values were consistantly lower for arm-crank than cycle ergometer exercise. In 1968, Nielsen (100) examined the core temperature responses of two subjects during arm-crank and cycle ergometer exercise. Rectal temperature values (mean of values obtained at four depths ranging from 12 to 27 cm past the anal sphincter) were found to be consistently lower during arm-crank than cycle exercise at a given absolute metabolic intensity. In contrast, esophageal temperature values appeared to be similar for the two exercise types at a given absolute metabolic rate (Figure 3-8).

[INSERT FIGURE 3-8 HERE]

In 1984, Sawka et al. (136) measured nine subjects' rectal temperature responses during arm-crank and cycle exercise performed at similar absolute and relative intensities. During the absolute intensity experiments, the subjects' steady-state rectal temperature values were the same for both exercise types. On the other hand, during the relative intensity experiments the subjects' rectal temperature values were lower during arm-crank than cycle exercise. Subsequently, in a different group of experiments, these investigators (133) found that their subjects' steady-state esophageal temperature values were the same for arm-crank and cycle exercise at the same absolute metabolic intensity. In 1987, Young et al. (173) had six subjects perform arm-crank and treadmill exercise while wearing protective clothing and receiving microclimate cooling. For the experiments that were comparable between the two exercise types, the steady-state rectal temperature values were not different at a given absolute metabolic intensity.

It seems clear that during upper body exercise the magnitude of core temperature elevation is not coupled to the relative exercise intensity. Several early studies (4,100) reported that rectal temperature values were lower during arm-crank than cycle exercise at a given absolute metabolic intensity. Later studies, however, reported no difference for rectal (136,172) or esophageal temperature (100,133) responses between the two exercise types at a given absolute metabolic intensity. The apparent discrepancy concerning the differences in rectal temperature responses may reflect methodological differences between the studies.

METABOLISM

Metabolic Rate

As defined by the heat balance equation (Chapter 1), body heat storage is equal to the difference between metabolic heat release and the heat loss to the environment. Physical exercise can increase total body metabolism by 5 to 15 times the resting rate in order to provide energy for skeletal muscle contraction. Depending upon the exercise task, between 70 to 100% of the metabolic rate is released as heat and needs to be dissipated to defend body temperature.

The effects of acute heat stress on man's ability to achieve maximal aerobic metabolic rates during exercise have been well studied. Most investigators find that maximal oxygen uptake is reduced in a hot as compared to a comfortable environment (72,119,122,137,139), but several investigators report no differences (118,168). For example, in one study (137) maximal oxygen uptake was 0.2 $\ell \cdot min^{-1}$ lower in a 49° C. as compared to a 21° C environment. The physiological mechanism responsible for the decreased maximal oxygen uptake is probably the diversion of blood to the cutaneous vasculature which could: (a) reduce the portion of the cardiac output perfusing the contracting musculature and/or, (b) cause a peripheral pooling of blood, reduce the central blood volume and thus reduce cardiac filling pressure and cardiac output.

Acute heat stress increases the metabolic rate for resting humans (26,27,28) and anesthetized dogs (138). Does heat stress alter an individual's metabolic requirements for performing a given submaximal exercise task? The answer to this question not only affects the calculation of the heat balance equation, but might also have implications for the nutritional requirements of individuals exposed to hot environments.

This latter concern stimulated Consolazio and colleagues (26,27) and later others to address the question of metabolic requirements for humans performing exercise in hot environments. Many investigators have found that to perform a given exercise task the absolute metabolic rate is greater in a hot than comfortable environment (26,27,38,46). Some investigators have reported the opposite results (19,108,168,173). The subject's state of heat acclimation does not account for whether they demonstrate an increased or decreased metabolic rate during exercise in the heat. We believe that this discrepancy can be explained and will attempt to do so in the following paragraphs. All of these studies, with the exception of Dimri et al. (38), only calculated the aerobic metabolic rate.

Dimri et al. (38) had six subjects perform cycle exercise at three power output levels in each of three environments. Figure 3-9 presents their subjects' total metabolic rate (bottom) and the percentage of this metabolic rate which is contributed by aerobic and anaerobic metabolic pathways. The anaerobic metabolism was calculated by measuring the post-exercise oxygen uptake that was in excess over resting baseline levels. Note that to perform exercise at a given power output, the total metabolic rate increased with the elevated ambient temperature. More importantly, the percentage of the total metabolic rate contributed by anaerobic metabolism also increased with the ambient temperature. The increase of anaerobic energy exceeded the increase of total metabolic rate during exercise at the elevated ambient temperatures. Therefore, if only the aerobic metabolic rate had been quantified, Dimri et al. (38) would have reported a decreased metabolic rate in the heat for performing exercise at a given power output. The investigations which reported a lower metabolic rate during exercise in the heat also reported increased plasma or muscle lactate levels (108,169,174) or an increased respiratory exchange ratio (19) which also suggests an increased anaerobic metabolism. Likewise, other investigators have reported that plasma lactate levels are greater during exercise in a hot as compared to a comfortable environment (37,38,46,91,114).

[INSERT FIGURE 3-9 HERE]

Skeletal Muscle Metabolism

Two investigations have examined the effects of environmental heat stress on skeletal muscle metabolism during exercise (46,174). Fink et al. (46) had six subjects perform 45 minutes of cycle exercise (70 to 85% of $\dot{V}O_2$max) in a cold (9° C, 55% rh) and a hot (41° C, 15% rh) environment. They found greater plasma lactate levels and increased muscle glycogen utilization during exercise in the heat. Also, muscle triglyceride utilization was reduced during exercise in the heat as compared to the cold. In addition, serum glucose concentration increased and serum triglyceride concentration decreased during exercise in the heat, compared to the opposite responses during exercise in the cold. During exercise in the heat, the increased muscle glycogen utilization was attributed to an increased anaerobic glycolysis resulting from local muscle hypoxia, which was caused by a reduced muscle blood flow. Since these investigators (46) did not perform the control experiments in a comfortable environment, the differences reported could be due partially to the effects of the cold exposure.

Young et al. (174) had thirteen subjects perform 30 minutes of cycle exercise (70% of $\dot{V}O_2$max) in a comfortable (20° C) and a hot (49° C) environment. They found that skeletal muscle and plasma lactate concentrations were greater during exercise in the heat. Interestingly, they also found a relationship (r=0.70) between the subjects' percent fast twitch fibers and the magnitude of muscle lactate increment in the hot environment (Figure 3-10). These investigators found no difference in muscle glycogen utilization between the two experimental conditions. Young et al. (174) speculated that during exercise in the heat, an alternative glycolytic substrate might

have been utilized, such as blood borne glucose. Rowell et al. (120) have shown a dramatic increase in hepatic glucose release into the blood during exercise in a hot compared to a comfortable environment. Such an increased release of hepatic glucose could account for the elevated serum glucose concentration reported in the hot environment by Fink et al. (46).

[INSERT FIGURE 3-10 HERE]

The data from Dimri et al. (38) and Young et al. (174) both support the concept of increased anaerobic metabolism during exercise in the heat. Much of the other support for this concept is based on the findings that, during exercise, the plasma lactate accumulation is greater in a hot than in a comfortable environment (37,38,46,91,114,169,174). However, any inference about metabolic effects at the muscle level from changes in plasma lactate are open to debate. Plasma lactate concentration reflects the balance between muscular production, efflux into the blood, and removal from the blood. Rowell et al. (120) have shown that compensatory vasoregulation reduces splanchnic removal of plasma lactate during exercise in the heat. Therefore, the greater blood lactate accumulation during exercise in the heat can be attributed, at least in part, to a redistribution of blood flow away from the splanchnic tissues.

The question remains as to what physiological mechanism(s) might be responsible for an increased anaerobic metabolism during exercise in the heat. As discussed, one possibility is the redistribution of blood to the cutaneous veins for heat dissipation; this may result in reduced perfusion of the active skeletal muscles and thus local tissue hypoxia (169). Hypoxia would be expected to cause a shift from aerobic to anaerobic pathways in order to provide energy for the skeletal muscle contraction. Another

possible explanation for increased anaerobic metabolism, is that acute heat exposure might increase the recruitment of fast-twitch motor units. Fast-twitch skeletal muscles derive a greater percentage of their total energy expenditure from anaerobic pathways (than slow-twitch fibers) regardless of their level of perfusion (135). In addition, fast-twitch fibers will expend a greater amount of energy to develop the same amount of tension as will slow-twitch fibers (135,159). A final possibility is that an elevated temperature may reduce the efficiency of skeletal muscle (18) and, therefore, increase the energy cost of contraction.

In conclusion, acute heat stress has marked effects on metabolism. During maximal exercise, an individual's maximal oxygen uptake is reduced by heat stress. During submaximal exercise, there is probably a greater total energy cost necessary in a hot as compared to a comfortable environment. The greater total energy cost may simply reflect the individual's discomfort in the heat, thus making more extraneous movements during exposure. On the other hand, biochemical and physiological mechanisms may also contribute to the greater total energy cost. Finally, it seems clear that there is a shift from aerobic to anaerobic metabolic pathways during exercise in the heat.

EVAPORATIVE HEAT LOSS

Sweat Gland and Evaporation

Figure 3-4 illustrates that when the ambient temperature increases, there is a greater dependence upon insensible (evaporative) heat loss to defend core temperature during exercise. For humans (unlike most animals) respiratory evaporative cooling is of little importance (88) and most evaporative heat loss occurs on the skin. This

provides the advantage of having a greater surface area available for evaporation. The eccrine glands secrete sweat onto the skin surface which causes evaporative cooling when it is converted from liquid to water vapor. The rate of evaporation is dependent upon the water vapor pressure gradient between the skin and the immediate environment (see Chapter 1): the larger the gradient, the greater the rate of evaporation. When a gram of sweat is converted into water vapor at 30° C, 2.43 kilojoules (kJ) of heat (the latent heat of evaporation) is absorbed in the process (161). Wenger (161) has shown that ambient temperature, relative humidity and solute content do not alter the latent heat of evaporation.

During cycle ergometer exercise it is not unusual for an individual to have a metabolic rate of 800 W ($\dot{V}O_2$ ~2.3 $\ell \cdot min^{-1}$). If the exercise performance is 20% efficient then the remaining 80% of the energy is converted to heat in the body, so that ~640 W (i.e., 0.64 $kJ \cdot s^{-1}$ or 38.4 $kJ \cdot min^{-1}$) needs to be dissipated to avoid body heat storage. The specific heat of body tissue is ~3.5 $kJ \cdot (kg \cdot ^{\circ} C)^{-1}$ (see Chapter 1) so that a 70 kg man has a heat capacity of 245 $kJ \cdot ^{\circ} C$. If such a man performed exercise in an environment that enabled only insensible heat exchange, and he did not sweat, the body temperature would increase by ~1.0° C every 6.3 minutes (Steps 2 and 3). Since the latent heat of evaporation for sweat is 2.43 kJ per gram, this man would need to evaporate ~16 grams of sweat per minute (Step 4) in order to achieve a steady-state body temperature. This amount of sweat output is not unreasonable, as values in excess of 30 grams per minute have been reported during exercise in the heat (140). The calculations describing the values discussed in this paragraph are provided below:

(Step 1) 640 W • 0.06 kJ $\cdot min^{-1}$ = 38.4 kJ $\cdot min^{-1}$ (Conversion of W to $kJ \cdot min^{-1}$)

(Step 2) 3.5 kJ $\cdot (kg \cdot ^{\circ} C)^{-1} \cdot$ 70 kg $\cdot 1^{\circ} C$ = 245 kJ

(Step 3) 245 kJ $\cdot (38.4 \text{ kJ} \cdot min)^{-1}$ = 6.3 min

(Step 4) 38.4 kJ $\cdot min^{-1}$ ÷ 2.43 kJ $\cdot g^{-1}$ = 15.8 grams of sweat per min

Thermoregulatory sweat in humans is secreted by about 1.6 to 4 million eccrine glands (75,125). The number of sweat glands per unit of skin surface area varies considerably between body regions (75). Eccrine sweat glands are most numerous on the sole of the foot and least numerous on the back (125). The fine structure of the eccrine sweat gland consists of the secretory coil, duct and skin pore. The secretory coil is composed of clear cells as well as dark mucoid and myoepithelial cells. The clear cells contain many mitochondria and are believed to be responsible for the secretion of sweat and electrolytes (125). The clear cells are probably dependent upon glucose as a substrate, but it is unclear whether the source is local glycogen stores or blood borne glucose (125). Sweating can cause glycogen depletion in the clear cells, but this apparently does not occur in heat acclimated individuals (125). The myoepithelial cells provide structural support for the secretory epithelium, but do not contribute directly to the expulsion of sweat as once believed (127).

The amount of sweat secreted by the eccrine gland is dependent upon the structure and function of the stimulated gland, as well as the sudomotor signal from the central nervous system. Sato and Sato (131) have recently excised human eccrine sweat glands and performed in vitro analyses of their structure and function. They found that individuals who were categorized as copious sweaters had larger eccrine glands. In addition, as the sweat gland size increased there were: (a) a greater maximal sweating rate per gland, (b) a greater sweating rate per unit tubular length or unit volume of secretory coil, and (c) a greater cholinergic sensitivity of the sweat gland. These findings emphasize the importance of eccrine gland size and cholinergic sensitivity on the thermoregulatory sweating response.

Control of the Sweat Gland

The sudomotor signal descends through the brain stem and spinal tracts to exit into the paravertebral ganglionic chain. The post-ganglionic sympathetic fibers which innervate the eccrine gland are nonmyelinated class C fibers that are primarily cholinergic. Vasoactive intestinal polypeptide (VIP) may also be released at the eccrine sweat gland (80). Lundberg et al. (80) have shown that VIP and acetylcholine are concomitantly released from the same post-ganglionic fibers innervating exocrine glands of the cat. Although both transmitters caused exocrine secretions, the VIP primarily acts by dilation of local vasculature to facilitate sweat function. For many years it has been known that eccrine sweat secretion and cutaneous vasodiltion are associated (79). It seems possible that the co-release of acetylcholine and VIP at the eccrine gland might explain this relationship (74,80). Finally, there are also some adrenergic alpha and beta receptors associated with eccrine sweat secretion (128,129,130).

Figure 3-11 represents the pattern of eccrine secretion, as measured by dew point hygrometry, during exercise in a warm environment. Note that sweat is normally secreted in a cyclic pattern, which reflects the pattern of action potentials transmitted via the sudomotor neurons and subsequent neuroglandular release (44,45). Thermoregulatory sweating can begin within a few seconds (155) to minutes (56) after starting muscular exercise. The increase in sweating rate closely parallels the increase in body temperature (96,123). As the sweating rate increases toward maximum levels, first there is a recruitment of sweat glands and then an increased sweat output per gland (65,111,126). Therefore, the sweat output for a given region of skin is dependent upon both the density of sweat glands as well as the sweat output per gland (65,96,111,126). Nadel et al. (96) have also demonstrated that individual regions of skin have different sweating responses for a given effector signal. Figure 3-12 presents the regional local sweating responses plotted against core temperature. Note that for a given core temperature, and thus thermal drive, the back and chest

have the greatest sweating rates. Conversely, the limbs will have relatively high sweating rates only after a substantial elevation in core temperature.

[INSERT FIGURES 3-11 AND 3-12 HERE]

Skin Temperature and Humidity

For a given effector signal to the eccrine gland, local skin temperature and skin wettedness influence the amount of sweat secreted. Bullard and co-workers (23) were the first to systematically evaluate the relationship between skin temperature and thermoregulatory sweating. They found that for an active eccrine gland, an elevated skin temperature induced a greater sweating rate. These results have since been verified and expanded upon by many other investigators (44,45,92,105,106). Figure 3-13 illustrates the effect of an increased mean skin temperature on the local sweating response for a given core temperature during muscular exercise.

[INSERT FIGURE 3-13 HERE]

Nadel et al. (92) conducted an elaborate study on the importance of skin temperature in the regulation of sweating. These investigators independently varied the mean skin temperature, core temperature, and local skin temperature, while measuring thigh sweating rate during rest, pre- and post-exercise. They concluded that: (a) at a constant skin temperature sweating rate was proportional to core temperature, (b) at a constant core temperature sweating rate was proportional to the mean skin temperature, and (c) at a given combination of core and mean skin temperatures local sweating was dependent on the local skin temperature. It was proposed that the local

skin temperature acted as a multiplier to the linear additive model of core and mean skin temperatures for the proportionate control system to determine local sweating rate.

The exact physiological mechanism responsible for mediation of the enhanced sweating response by elevated local skin temperatures, is unclear. It is clear, however, that there are several factors which in combination contribute to enhance sweating. These factors include increased neurotransmitter release and increased glandular responsiveness. Some investigators believe that local skin heating results in a greater release of neurotransmitter substance for a given sudomotor signal arriving at the eccrine sweat gland (23,45,46,92,105,106). The greater neurotransmitter release would, therefore, stimulate greater sweat production and release. Ogawa and colleagues (105,106) have provided evidence that local heating increases the eccrine glands responsiveness to a given amount of neurotransmitter substance. It is unknown if this increased glandular responsiveness is receptor mediated or reflects increased cellular metabolism within the secretory coil.

Remember, the rate of sweat evaporation is dependent upon the water vapor pressure gradient between the skin and the environment. Unevaporated sweat lying on the skin surface results in a reduced sweat output (25,56,98). The action of increased skin wettedness causing sweat suppression is called hidromeiosis. The mechanism(s) responsible for hidromeiosis is/are unknown, although several hypotheses have been proposed. Peiss et al. (107) suggest that the wetted skin might cause the stratum corneum to swell and cause mechanical obstruction of the sweat duct. This idea was systematically tested by Brown and Sargent (22) who supported the concept that the stratum corneum was responsible for hidromeisis. Nadel and Stolwijk (98) have hypothesized that excessive skin wettedness might reduce, via dilution, the osmotic gradient along the sweat duct, thus reducing sweating rate. They proposed that sweat may follow an osmotic gradient (from the solutes left behind by evaporated sweat)

from the proximal duct to the skin surface. Candas et al. (25) have suggested that hidromeiosis may result, by negative feedback, from locally wet skin which reduced local sweat output. All of the above hypotheses, with the exception of the first, are tentative and more research is merited concerning skin wettedness and the mechanisms of sweat suppression. Interestingly, if the environmental evaporative capacity is increased and subsequently dries the skin, the sweating rate will be potentiated (98,150). For example, Nadel and Stolwijk (98) demonstrated that when individuals were exposed to increased air motion, they had an elevated sweating rate at a given core temperature during exercise in a warm-dry environment.

Several investigators (154,171) have proposed that the eccrine sweat glands may fatigue during prolonged periods of high sweat output; the notion being that the fatigued eccrine gland is unable to respond to a constant or even increasing thermoregulatory drive for sweating (22). The evidence supporting the idea of sweat gland fatigue is that: (a) during prolonged (3-5 h) periods of heat exposure, both the steady-state (53,154,171) and maximal (171) sweating rates are reduced and, (b) that these "fatigued" eccrine glands demonstrate reduced responsiveness to the subdermal injection of a sudorific agonist (154). Examination of these studies, however, can lead one to conclude that excessive skin wettedness causing hidromeiosis and/or dehydration (Chapter 6) may account for much of the so-called glandular fatigue.

Brown and Sargent (22) conducted a series of experiments to determine if hidromeiosis could be responsible for the proposed eccrine gland fatigue. They found that during prolonged (4-8 h) heat exposure, the decline in sweat output could be reversed if the subject were simply moved from a moist-heat to a dry-heat. Brown and Sargent (22) then subdermally injected a sudorific agonist into the skin and measured the local sweating responses both before and after a 4-6 h walk in the heat. These injections were made in areas of intact skin and in areas of skin that were

stripped of the stratum corneum. They found that total body sweating rate decreased after several hours and continued to decrease throughout the duration of the exercise-heat exposure. During this period the local sweating rates decreased in the intact skin but remained more constant for the stripped skin. Likewise, injection of the sudorific agonist demonstrated a reduced sweat responsiveness in the intact skin but no change in sweat responsiveness in the stripped skin. They concluded that excessively wetted skin results in swelling of the stratum corneum which obstructs the sweat duct and causes the so-called sweat gland fatigue.

The findings of Brown and Sargent (22) do not discount the possibility that eccrine sweat glands may fatigue. Future experiments that examine sweat gland fatigue will need to control for skin wettedness and hydration levels. Finally, in a recent study where subjects exercised in a hot-dry environment (35° C, 20% rh) for twelve hours while euhydration was maintained, no differences in the total body sweating rate were found between the first and twelth hour of exercise (76). These experiments elicited moderate (~9g•min^{-1}) sweating rates, and it would be interesting to see if similar results were found at higher sweating rates.

In conclusion, during exercise in hot environments, the evaporation of eccrine sweat is an important avenue of heat exchange. The eccrine glands are primarily controlled by the thermoregulatory centers via sympathetic cholinergic nerves. For humans, core temperature provides the primary thermoregulatory drive for sweating; however, skin temperature and skin wettedness modify this response.

SKIN CIRCULATION AND HEAT TRANSFER

Control of Skin Blood Flow

Skin blood flow carries heat by convection between the deep body tissues and the skin. When the environment is cool enough not to elicit sweating, raising skin blood flow brings skin temperature nearer to blood temperature, and lowering skin blood flow brings skin temperature nearer to ambient temperature. In such an environment, the body is able to control sensible, or dry, heat loss by varying skin blood flow, and thus skin temperature. If the heat stress is great enough that sensible heat loss alone is not enough to maintain heat balance, and sweating begins. Skin blood flow continues to increase with increasing heat stress, but has only a minor effect on skin temperature and sensible heat exchange, and serves primarily to deliver to the skin the heat that is being removed by sweat evaporation. Skin blood flow and sweating thus work in tandem to dissipate heat under such conditions.

The skin circulation is affected by temperature in two ways: local skin temperature affects the vascular smooth muscle directly, as will be discussed later in this section; and temperatures in the core and of the skin elsewhere on the body affect skin blood flow by reflexes operating through the sympathetic nervous system. Blood flow in much of the human skin is under a dual vasomotor control (49, 117). During cold exposure (and also with certain non-thermal reflexes—see Figure 3-2), skin blood flow is reduced through the action of vasoconstrictor fibers. These fibers presumably are adrenergic, since their action is blocked by bretylium (10). In the hands, feet, lips, ears, and nose, these seem to be the predominant vasomotor innervation, and the vasodilation that occurs in these regions during heat exposure is largely a passive result of the withdrawal of vasoconstrictor activity. Over most of the skin area, however, vasoconstrictor activity is already minimal under conditions of thermal comfort, and vasodilation during heat exposure depends on intact sympathetic innervation, since it can be prevented or reversed by regional nerve block. Since it depends on the action of neural signals, such vasodilation is sometimes referred to as active vasodilation.

Both active vasoconstriction and active vasodilation play a major part in controlling skin blood flow of the upper arm, forearm, thigh, and calf (11). However, active vasoconstriction is believed not to have a major role in controlling skin blood on the trunk or on most of the head (12,50).

Although active vasodilation occurs in limited skin areas (mostly paws and tail) in many animal species, human beings seem to be unique in the intensity of the response and the amount of skin involved (116). The vasoactive agonist responsible for active cutaneous vasodilation in man has not yet been identified, but some investigators have proposed that vasodilation is mediated through the action of sweat glands. It has been proposed that sweat glands, when stimulated to secrete sweat, release bradykinin, a powerful vasodilator, into the interstitial space of the skin, where it acts on the arterioles of the skin. This hypothesis now seems doubtful, and Rowell (116) has summarized the arguments against it. Nevertheless, it is possible that active vasodilation depends on sweat gland activity through some other vasodilator, such as release of VIP. Evidence suggesting a relationship of active vasodilation to sweating includes observations on some patients with anhidrotic ectodermal dysplasia, a rare hereditary condition in which sweat glands are absent. Although these patients had normal vasoconstrictor activity in their forearms, and their forearm skin dilated normally in response to local heating, no active vasodilation occurred in response to whole-body heating (16). Since patients with anhidrotic ectodermal dysplasia usually have normal autonomic function, it is tempting to conclude from these experiments that active vasodilation is secondary to some action of the sweat glands. Other explanations are possible, however, such as that both the sweat glands and the nerve endings responsible for active vasodilation fail to develop during embryogenesis in this disorder.

Besides contributing to the reflex control of skin blood flow, skin temperature also affects skin blood flow through direct actions on the blood vessels themselves (20,21).

as shown in Figure 3-14. Local temperature changes act on skin blood vessels in at least two ways. First, local cooling potentiates, and heating weakens, the contractile response of vascular smooth muscle to norepinephrine and other constrictor agonists, apparently by changing the affinity of α_2 adrenoceptors for these agonists (70). Second, in human forearm skin (and presumably in other skin regions where active vasodilation occurs) local heating causes vasodilation, and local cooling causes vasoconstriction, even in the absence of nervous signals (167). Through this direct vasodilator effect, local heating increases skin blood flow so much that most of the heat delivered to the skin is carried away by the blood and little is conducted to the deeper tissues. This response reinforces thermoregulatory vasomotor responses, and also tends to protect the skin from heat injury.

[INSERT FIGURE 3-14 HERE]

Heat Transfer from Skin Blood Flow

Blood has a volume specific heat of 3.85 $kJ \cdot \ell^{-1} \cdot {}^{0}C^{-1}$ (0.92 $kcal \cdot \ell^{-1} \cdot {}^{0}C^{-1}$) so that if a liter of blood at 37° flows through the skin and returns to the body core at 36° C, the body loses 3.85 kJ of heat. The muscle blood flow necessary to sustain aerobic metabolism depends on the O_2 content of the arterial blood, which is seldom much above 200 ml O_2 per liter. The chemical energy released when 200 ml O_2 is consumed depends somewhat on the substrate being oxidized, but is about 4.2 kJ. Thus, even if the muscle uses all of the oxygen in the arterial blood, the muscle needs to be only 1.09° C warmer than the arterial blood in order for the blood perfusing it to remove the heat that it is producing. Therefore, the blood flow needed to sustain aerobic metabolism provides amply for removing heat from exercising muscle. The rate

of core-to-skin heat transfer by skin blood flow depends on the rate of skin blood flow (SkBF) and on the temperature difference between blood leaving the core on its way to the skin and blood returning to the core from the skin. In the most efficient case, this temperature difference is equal to the difference between core and skin temperature (T_c and T_{sk}), and core-to-skin heat transfer (HF) is:

$$HF = SkBF \cdot (T_c - T_{sk}) \cdot 3.85 kJ \cdot \ell^{-1} \cdot {}^{\circ}C^{-1} \quad (4)$$

In practice, especially in a cool subject, heat transfer is less efficient. As the warm blood leaves the body core via the arteries, it flows through superficial tissues that are cooler than the core but warmer than the skin, and as it flows through these tissues, the blood gives up some of its heat to them. The blood that enters the skin is thus cooler than the core, so that heat loss from the blood to the skin is less than that given by equation 4. Some of the heat that the blood has lost on its way to the skin eventually finds its way to the skin by conduction, but some of this heat is returned to the cooled venous blood on its way back to the core, as it passes through the same layers of tissue through which it passed on its way outward. One may think of this heat as having taken a "short circuit" from the arteries to the veins, without having passed through the skin. This process, the exchange of heat between the blood and the tissues through which it passes on the way between core and skin, thus has the effect of reducing convective heat transfer by the blood, or alternatively of requiring more blood flow to cause a given heat transfer than would be required in the most efficient case assumed in equation 4.

The difference between actual core-to-skin heat exchange and that predicted by equation 4 is subject to physiological control in the limbs. To explain this, it is first necessary to describe the dual venous drainage of the limbs, consisting of deep veins,

which ordinarily drain blood mainly from the muscles; and superficial veins, which lie in the skin and subcutaneous tissue and ordinarily drain blood mainly from the skin. However, the deep and superficial veins are connected by many penetrating veins, so that potentially blood from anywhere in the limbs can return to the heart through either deep or superficial veins. The deep veins have a relatively poor sympathetic innervation (157), but the superficial veins have a rich sympathetic innervation, constricting when the skin or body core is cooled and dilating when the skin or core is warmed (158,165). In a cool subject, therefore, most blood from the limbs returns to the core via the deep veins. Since these veins and the major limb arteries lie adjacent to each other for a considerable part of their length, some of the heat from the arterial blood flow is conducted to the cooler venous blood and returned to the body core, and so is conserved. Such heat exchange between parallel streams moving in opposite directions is called "counter-current" heat exchange. In a warm subject, however, counter-current heat exchange is no longer advantageous, and it is considerably diminished by dilation of the large superficial veins, which allows much of the venous blood to return to the core along paths far removed from the major limb arteries. Not only does dilation of the larger superficial veins increase core-to-skin heat transfer by minimizing counter-current heat exchange, but also dilation of the smaller superficial veins may further improve heat transfer by increasing the time that the blood remains in the skin, and there is evidence that the degree of venous filling affects heat transfer to the skin (55).

Venous occlusion plethysmography is the most quantitative non-invasive method for measuring blood flow. However, it can be used only on the extremities, and the requirement that the part being studied be elevated above the heart to provide venous drainage limits its use to the hand and forearm in most situations. Since muscle blood flow is generally not influenced by thermal reflexes (40), changes in hand or

forearm blood flow can be taken to primarily represent changes in skin blood flow. Measurements of heat flow, either through the skin as a whole or through a restricted area, are sometimes divided by the temperature difference between core and skin to give a thermal conductance. Even in the absence of blood flow, there will be some heat flow due to the thermal conductivity of the tissues themselves, but after the contribution of this heat flow to thermal conductance is subtracted, the remainder can be used as an index of skin blood flow. However, such measurements only give values for the minimum blood flow rate which could produce the observed heat flow. For reasons discussed earlier, the actual blood flow may be, and probably usually is, greater; but accurate data on the relation between heat flow and blood flow are not available. Furthermore, if this method is used to provide an index of blood flow to the entire skin, potentially serious errors may be introduced by regional variation in skin temperature. Given the lack of a better practical index, forearm blood flow is often used as an index of skin blood flow, since both types of vasomotor control, constrictor and dilator, are represented in forearm skin. However, very little information is available concerning how good an index forearm blood flow actually is under different conditions.

CARDIOVASCULAR CONSEQUENCES OF THERMOREGULATORY INCREASES IN SKIN BLOOD FLOW

Thermoregulatory Skin Blood Flow Responses to Exercise-Heat Stress

Although skin temperature is higher the warmer the environment, core temperature is relatively unaffected by environmental temperature over a fairly wide range, as discussed above. Thus in a warm environment the core-to-skin thermal gradient is

relatively narrow, and skin blood flow increases in response to the high skin temperature, so as to achieve core-to-skin heat transfer sufficient for thermal balance (eq. 4). During exercise, metabolic rate and heat production may be ten or more times their levels at rest. A healthy but not athletic, young 70-kg man may have a metabolic rate at rest of 80 W and a $\dot{V}O_2max$ of 3.5 $\ell \cdot min^{-1}$, which corresponds to a metabolic rate of 1215 W. Even though core temperature rises during exercise and thus widens the core-to-skin temperature gradient somewhat, this effect on core-to-skin heat transfer is far too little to match the increase in metabolic heat production. Besides widening the core-to-skin temperature gradient, the rising core temperature elicits reflex increases in skin blood flow, which eventually produce a rate of core-to-skin heat transfer sufficient to re-establish heat balance.

During exercise in the heat the narrow core-to-skin temperature gradient and the high rate of metabolic heat production that needs to be dissipated combine to require high levels of thermoregulatory skin blood flow. It is not possible to measure whole-body skin blood flow directly, but we can make an approximate estimate of skin blood flow during exercise from eq. 4. Let us consider again the man performing cycle exercise at a metabolic rate of 800 W and 20% efficiency, for a rate of heat production of 640 W. If at steady state his mean skin temperature is 35.5° C and core temperature is 39° C, and if we ignore respiratory heat loss, eq. 2 becomes 640 W $= SkBF \cdot (3.5^{\circ} C) \cdot 3.85 kJ \cdot \ell^{-1} \cdot {}^{\circ} C^{-1}$. Since 1 W = 0.001 $kJ \cdot s^{-1}$ = 0.06 $kJ \cdot min^{-1}$, solving for SkBF we have SkBF = 2.8 $\ell \cdot min^{-1}$. This is a rather crude estimate of skin blood flow, and should be taken only as an approximate figure. Although the man in this example is under considerable thermal stress, his estimated skin blood flow is well below the maximum obtainable. Rowell estimated the maximum skin blood flow obtainable by the combined effects, local and reflex, of heating the skin and body core. He computed skin blood flow of heated resting subjects as the sum of increases

in cardiac output and decreases in splanchnic, renal, and muscle blood flow during heating (115). At the end of heating, core temperature was 39.1 ^{o}C and still rising at an undiminished rate, skin temperature was 40.5^{o}C, and computed skin blood flow was 7.8 $\ell \cdot min^{-1}$ and had nearly stopped rising, and so was probably near the maximum obtainable by skin and whole-body heating. Such a high skin blood flow may be incompatible with the muscle blood flow required during exercise, and at any event is unrepresentatively high for exercise because of the effects of the high skin temperature.

Circulatory Strain During Exercise-Heat Stress

It is evident from the foregoing examples that during exercise-heat stress thermoregulatory skin blood flow, though not precisely known, may be substantial. The higher skin blood flow will, in general, result in a higher cardiac output, and one might expect the increased work of the heart in pumping this blood to be the major source of cardiovascular strain associated with heat stress. The work of the heart in providing the skin blood flow necessary for thermoregulation in the heat does, in fact, impose a substantial cardiac strain on patients with severe cardiac disease (24). In healthy subjects, however, the cardiovascular strain associated with heat stress results mostly from reduced cardiac filling and stroke volume (Fig. 3-15), which require a higher heart rate to maintain cardiac output. This occurs because the venous bed of the skin is large and compliant and, moreover, dilates reflexly during heat stress (Fig. 3-13; 116). Therefore, as skin blood flow increases, the blood vessels of the skin become engorged and rather large volumes of blood pool in the skin, thus displacing blood from the thorax and reducing central blood volume and cardiac filling (Fig. 3-16) (see refs. 116, 117). Since about 70% of the blood volume in an upright human being is below heart level (116), it is in the upright posture that the cardiovascular effects of

this blood pooling are greatest. Thus, for example, stroke volume during exercise is lower in the upright than in the supine posture, and this difference between postures is greatest in the heat (112).

[INSERT FIGURES 3-15 AND 3-16 HERE]

In addition, exercise and heat stress affect plasma volume in two ways. First, they cause fluid movements between plasma and tissues (Chapter 6). These fluid movements occur rather quickly, well before any substantial losses of fluid have occurred by sweating (Fig. 3-15). The overall magnitude and direction of these fluid movements depends on a number of factors, such as temperature, exercise type, hydration level, and status of heat acclimation, so that the overall effect may be either to increase or to decrease plasma volume, and thus to alleviate or to aggravate the circulatory effects of pooling of blood in the skin. Second, much fluid is lost by sweating during exercise and heat stress. Since the main solute in sweat is sodium chloride, the water in the sweat will be lost disproportionately at the expense of extracellular fluid, including plasma, to the extent that the body's content of sodium ion is depleted. The disproportionate loss from extracellular fluid will be minimized if the sweat is dilute, as is the case with subjects well acclimatized to heat. If the water and salt lost by sweating are not replaced, plasma and extracellular fluid volumes will be progressively reduced during exercise-heat stress.

Compensatory Responses to Maintain Cardiovascular Homeostasis During Exercise-Heat Stress

In the face of peripheral pooling of blood and possible decreases in plasma volume, several reflex adjustments occur that help to maintain cardiac filling, cardiac output, and arterial pressure during exercise and heat stress. Splanchnic and renal blood flows are reduced during exercise (115). The reduction in splanchnic blood flow is in proportion to relative exercise intensity (i.e., as a percent of $\dot{V}O_2max$), and presumably the same is true of renal blood flow (115). These blood flows also undergo a graded and progressive reduction in subjects heated at rest; and in the splanchnic bed, at least, the vasoconstrictor effects of temperature and of exercise appear to be additive, so that at any exercise intensity the reduction in splanchnic blood flow is greater at a higher skin temperature (115). The splanchnic vascular bed, and the hepatic vascular bed into which it drains via the hepatic portal vein, are very compliant, so that a reduction in splanchnic blood flow is followed by a reduction in the amount of blood pooled in these beds (Figure 3-16). A substantial volume of blood can thus be mobilized from these beds to help maintain cardiac filling during exercise and heat stress.

Since skin blood flow can reach such high levels during exercise-heat stress, and since high skin blood flow may produce substantial peripheral pooling of blood, one might ask whether exercise affects skin blood flow in the same way that it affects renal and splanchnic blood flow. Since exercise quickly warms the body, and since thermal factors play such a large role in control of skin blood flow, especially during exercise in the heat, it is useful to re-state the question so as to separate thermal from non-thermal influences on skin blood flow. We shall do this by asking whether exercise affects the relation of skin blood flow to core and skin temperatures and, if so, whether the effect of exercise is proportionate to relative exercise intensity (percent $\dot{V}O_2max$). Under conditions of high cardiovascular strain, such as during very intense exercise or at high skin temperatures, skin blood flow at a given core and skin

temperature is known to be reduced during exercise (115, 117). However, at moderate skin temperatures, the relation of skin blood flow to core and skin temperatures is independent of exercise intensity over a range from mild to fairly intense exercise (69, 165; see Reference 161 for further discussion). A more recent study has confirmed these findings, and extended them by showing that at a sufficiently high exercise intensity, skin blood flow is lower than would be expected from the levels of core and skin temperature, and this difference between actual and expected blood flows becomes greater as exercise intensity increases further (151). Thus in the service of cardiovascular homeostasis, the body begins to reduce splanchnic and renal blood flows at fairly low levels of cardiovascular strain, while sparing thermoregulatory skin blood flow, which begins to be compromised only at fairly high levels of cardiovascular strain.

[INSERT FIGURE 3-17 HERE]

Skin blood flow also is affected, especially during exercise in the heat, by other reflexes which function to maintain cardiac output and blood pressure, and which are elicited in situations in which cardiac filling is compromised. Thus during cycle exercise forearm vasodilation begins at a higher core temperature in the upright than in the supine posture, and this postural difference is greater at higher ambient temperatures, in which there presumably is more peripheral pooling (112). Likewise, during cycle exercise in the heat, forearm vasodilation begins at a higher core temperature if blood volume has been reduced before the start of exercise by a 4-day course of diuretics (94) or by acute blood withdrawal (47). However, the core temperature threshold for forearm vasodilation seems not to be affected by acute expansion of blood volume before the start of exercise (47). In some experiments, a break has been observed in the relation of forearm blood flow to core temperature (47,

93, 94) such that the slope of the relation is much less past the break (Figure 3-17). Moreover, such breaks have sometimes been seen to occur at a lower forearm blood flow under conditions of greater cardiovascular strain (e.g., during exercise with reduced blood volume) (94). These breaks are not intrinsic to the thermal control of skin blood flow, since they can be removed by having the subject exercise in the supine position (Figure 3-17), which favors return of blood to the heart, and cardiac filling. These breaks may represent the recruitment of a compensatory vasomotor adjustment to limit cutaneous vascular pooling.

Lower body negative pressure (LBNP) is an experimental technique in which the lower half of the body is enclosed in a rigid box to which negative pressure, to about 90 torr below atmospheric, is applied, to cause pooling of blood in the lower part of the body. Application of LBNP reduces central blood volume and central venous pressure, and unloads the cardiopulmonary baroreceptors. With low levels of LBNP, there is little effect on arterial pressure, but mean arterial pressure is reduced with higher levels of LBNP. Forearm blood flow is reduced during application of LBNP, even at levels low enough that arterial pressure is not affected, so that the response of forearm blood flow represents vasoconstriction in response to the reduction in central venous pressure (116). If forearm blood flow has previously been raised by heating the subject, the decrease in blood flow in response to LBNP can be much greater than in a thermoneutral subject (116). Intravascular pressures were not measured in the studies described in the previous paragraph, and impairment of cardiac filling was inferred from reductions in stroke volume. However, the changes in the control of forearm blood flow that were observed in conditions in which stroke volume was reduced were probably mediated by the cardiopulmonary baroreceptors, and probably represent the same reflex as that elicited by applying LBNP.

At a given level of skin blood flow, the volume of blood that pools in the cutaneous vessels depends on the compliance of the cutaneous veins, and thus can be reduced by constriction of these veins. With light to moderate exercise, the cutaneous veins constrict at the beginning of exercise, but relax within a few minutes (116), perhaps in response to the increase in core temperature. However, cutaneous venous volume seems to be more sensitive to exercise than is skin blood flow (163), and the venoconstriction during more intense exercise is sustained (48, 59).

All of the adjustments discussed above help to maintain cardiac filling and cardiac output during exercise in the heat. Most of this cardiac output goes to supply exercising muscle, but it is not known whether muscle blood flow is unaffected by heat stress, and quantitative measurements of blood flow through exercising muscle are not available. Rowell (115) has summarized much of the evidence bearing indirectly on this point, but this evidence is insufficient for a clear case either way. Some other recent evidence, though also somewhat indirect, suggests that heat stress may, in fact, compromise muscle blood flow, since the initial rise in blood lactate during exercise is more rapid during exercise at an ambient temperature of 35° C than at 25° C (91).

CONCLUSION

Human body temperature is regulated by a proportionate control system. It is unclear, however, which internal body temperature is regulated by the control of the thermoregulatory effector responses of skin blood flow and sweating. The core temperature increases during exercise as a result of a "load error" and not a change in the regulated set-point temperature. During exercise the magnitude of core temperature elevation at steady-state is proportional to the metabolic rate and is largely independent of the environmental condition. However, dependent upon the environmental conditions

the relative contributions of sensible (radiative and convective) and insensible (evaporative) heat exchange to the total heat loss will vary. The hotter the environment the greater the dependence on insensible heat loss.

During exercise in the heat, the primary problem is to simultaneously provide the cardiovascular support to maintain the metabolism for skeletal muscle contraction and to dissipate the associated heat release. In hot environments, the core to skin temperature gradient is reduced to skin blood flow needs to be relatively high (compared to cooler environments) to achieve heat transfer sufficient for thermal balance. In addition, sweat secretion can result in a reduced plasma (by dehydration) and thus blood volume. Both high skin blood flow and reduced plasma volume can reduce cardiac filling and perhaps cardiac output during exercise in the heat. As a result syncope or reduced exercise performance will occur.

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FIGURE LEGENDS

Figure 3-1. Differences between the elevation of core temperature in fever and the elevation of core temperature in exercise. In fever, the primary event is a rise in set point temperature (T_{set}), which initially causes a negative load error or error signal (-e). Heat-dissipating responses are inhibited and heat production is stimulated until T_c rises enough to correct the error signal and establish a new thermal balance in which heat production and heat loss are near (or slightly above) their values before the fever. During exercise T_c rises because of the increase in heat production. T_{set} is unchanged, and heat-dissipating are elicited as T_c rises. T_c continues to rise until it is high enough that the error signal (e) elicits heat loss responses at a rate sufficient to match heat production, and produce a new thermal balance. Redrawn from Reference 143.

Figure 3-2. Schematic model of the control of human thermoregulatory effector responses.

Figure 3-3. Rectal and esophageal temperature responses to rest and exercise in the heat.

Figure 3-4. The steady-state heat exchange data for one subject performing constant intensity exercise in a variety of ambient temperatures. Redrawn from Reference 103.

Figure 3-5. Relationship of steady-state core temperature responses during exercise at three metabolic rates to the environmental conditions. Redrawn from Reference 78.

Figure 3-6. Relationship of steady-state core temperature responses during exercise to the absolute and relative metabolic rates. Redrawn from Reference 124.

Figure 3-7. Relationship of steady-state core temperature responses during exercise to the relative exercise intensity and the ambient temperature. Left panel redrawn from Reference 33.

Figure 3-8. Relationship of steady-state esophageal temperature responses to arm-crank and cycle exercise at a given absolute metabolic rate. Redrawn from Reference 100.

Figure 3-9. The total metabolic rate and percentage contribution of aerobic and anaerobic metabolism during exercise at different ambient temperatures. Redrawn from Reference 38.

Figure 3-10. The relationship between the subject's percentage of fast-twitch muscle fibers and the magnitude of muscle lactate increment in a hot compared to comfortable environment during exercise. Redrawn from Reference 173.

Figure 3-11. The pattern of eccrine secretion, as measured by dew point hygrometry, during exercise in the heat.

Figure 3-12. The regional sweating rate responses plotted against core temperature. Redrawn from Reference 96.

Figure 3-13. The relations of back sweat rate (left), forearm blood flow (center), and forearm venous volume (right) to esophageal and mean skin temperatures (T_{es} and T_{sk}). Sweating data are from four subjects performing cycle exercise at an O_2 consumption rate of 1.6 ℓ/min. Measurements of forearm blood flow and forearm venous volume are from one subject each. Forearm temperature was kept at 36.8° C during measurements of blood flow, and at 35.1° C during measurements of forearm volume to eliminate a difference in local temperature between experiments at different ambiant temperatures. Local temperature was not controlled independently during measurements of sweating, so that the difference between conditions includes a small effect of local skin temperature, appearing as a difference in slope. (Left panel drawn from data of Reference 133; center panel modified from Reference 165; right panel modified from Reference 163).

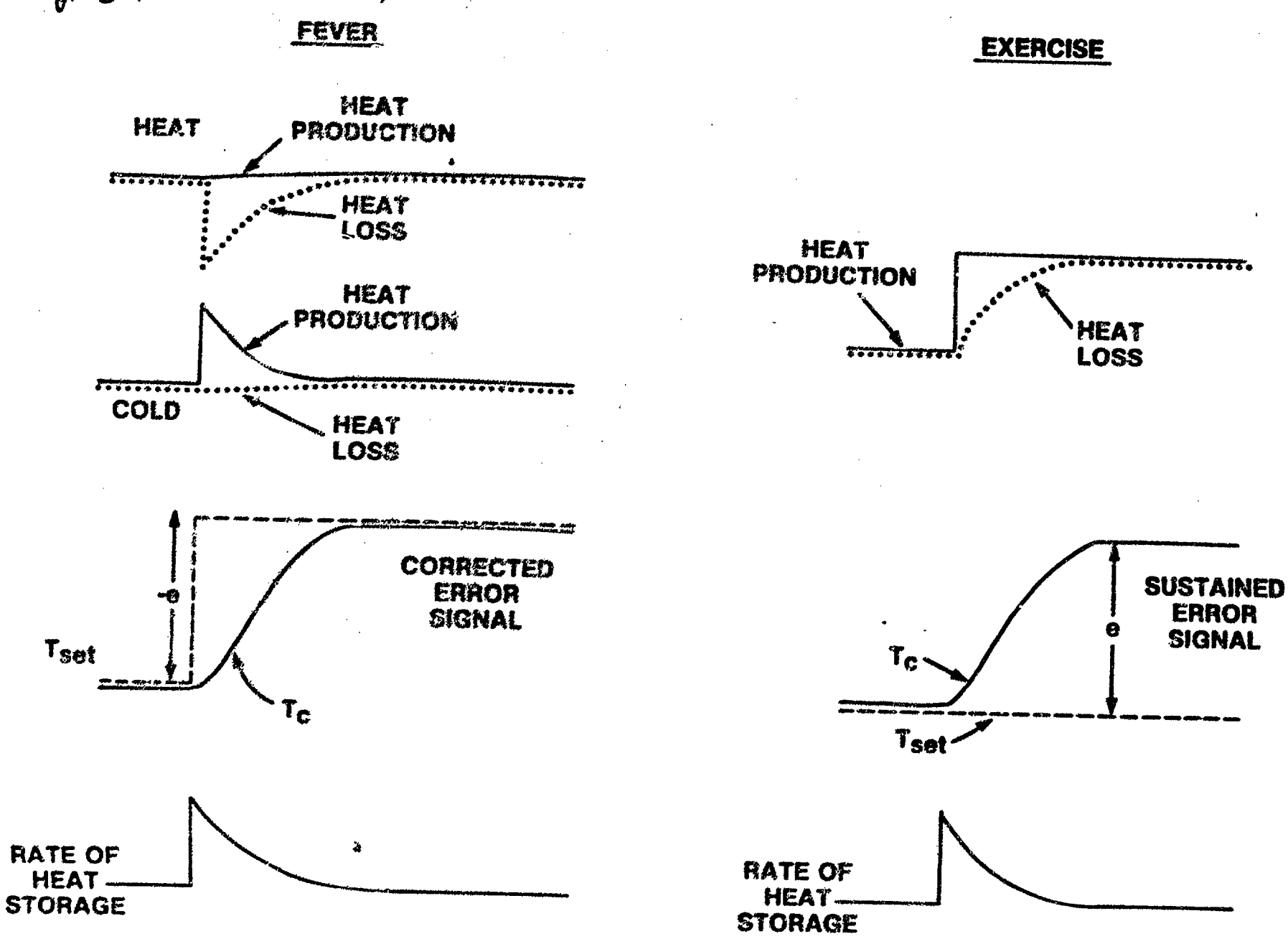
Figure 3-14. Effect of local temperature on forearm blood flow during cycle exercise. The skin of the experimental forearm was either heated to 39° C (open circles) or cooled to 27° C (filled circles), and the skin of the control forearm was maintained always at 33° C. Blood flow in the experimental forearm is plotted against blood flow measured simultaneously in the control forearm. If there was no effect of local temperature, plotted points would lie on the line of identity. Drawn from data reported in Reference 162.

Figure 3-15. Thermal and circulatory responses of one subject during cycle exercise at 70% $\dot{V}O_2$max in ambient temperatures of 20 and 35° C, showing (from top) esophageal and mean skin temperatures, cardiac output, stroke volume, percent change in plasma volume, and forearm blood flow. Drawn from data taken from Reference 93.

Figure 3-16. Schematic diagram of the effects of skin vasodilation on the thoracic reservoirs from which the ventricles are filled, and also effects of compensatory vasomotor adjustments in the splanchnic circulation. Redrawn from References 115, 116.

Figure 3-17. Relation of forearm blood flow to esophageal temperature during exercise in supine and seated positions in an ambient temperature of 36° C. Data points are averaged from duplicate experiments on one subject. Redrawn from Reference 93.

Fig. 3-1


FEVER
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RATE OF HEAT STORAGE
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HEAT PRODUCTION
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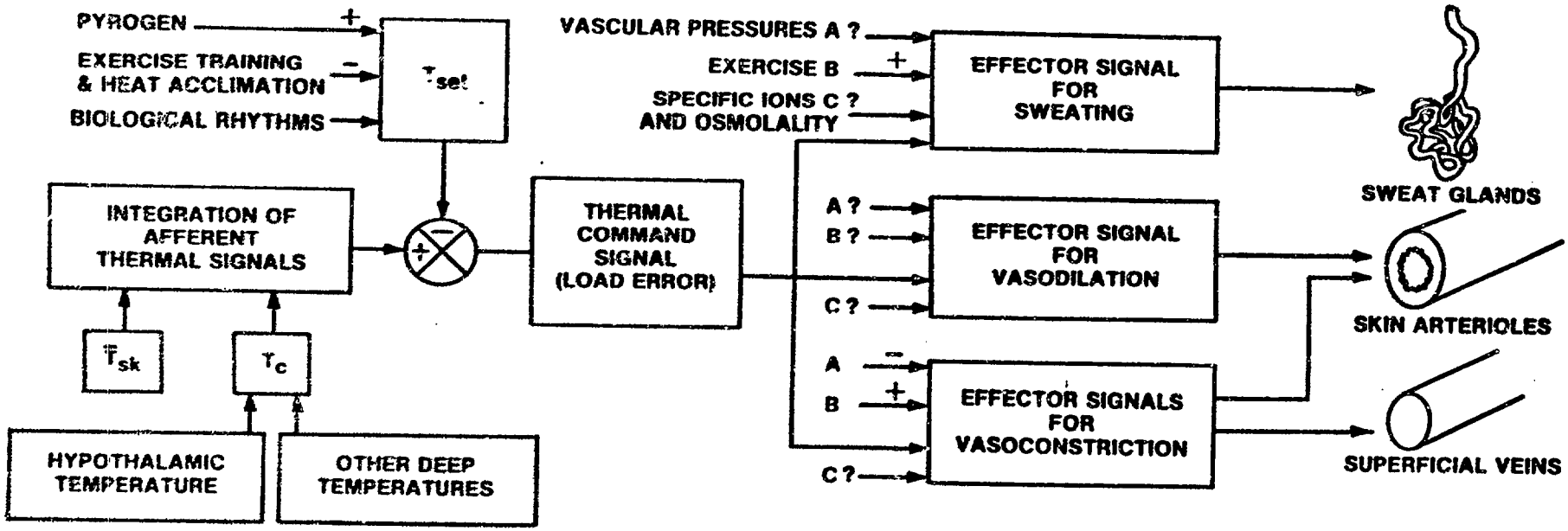
SUSTAINED ERROR SIGNAL
T_c
T_{set}
RATE OF HEAT STORAGE

Fig. 3-2


PYROGEN
+
EXERCISE TRAINING
& HEAT ACCLIMATION
−
BIOLOGICAL RHYTHMS
T_{set}
INTEGRATION OF
AFFERENT
THERMAL SIGNALS
T_{sk}
T_c
HYPOTHALAMIC
TEMPERATURE
OTHER DEEP
TEMPERATURES
THERMAL
COMMAND
SIGNAL
(LOAD ERROR)
VASCULAR PRESSURES A ?
EXERCISE B
+
SPECIFIC IONS C ?
AND OSMOLALITY
EFFECTOR SIGNAL
FOR
SWEATING
A ?
B ?
C ?
EFFECTOR SIGNAL
FOR
VASODILATION
A
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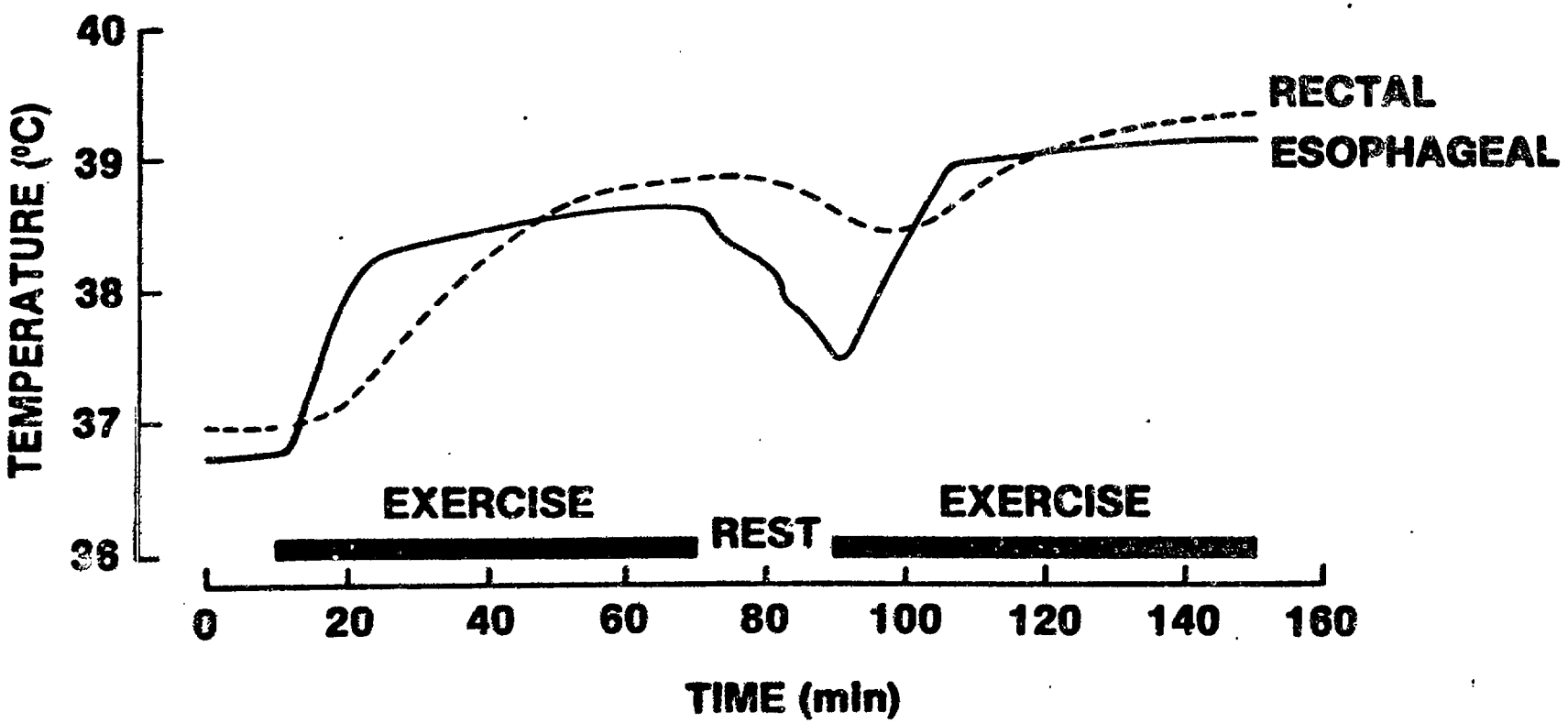
EFFECTOR SIGNALS
FOR
VASOCONSTRICTION
SWEAT GLANDS
SKIN ARTERIOLES
SUPERFICIAL VEINS

Fig. 3-3


40
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TEMPERATURE (°C)
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EXERCISE
0
20
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80
100
120
140
160
TIME (min)

Fig. 3-4

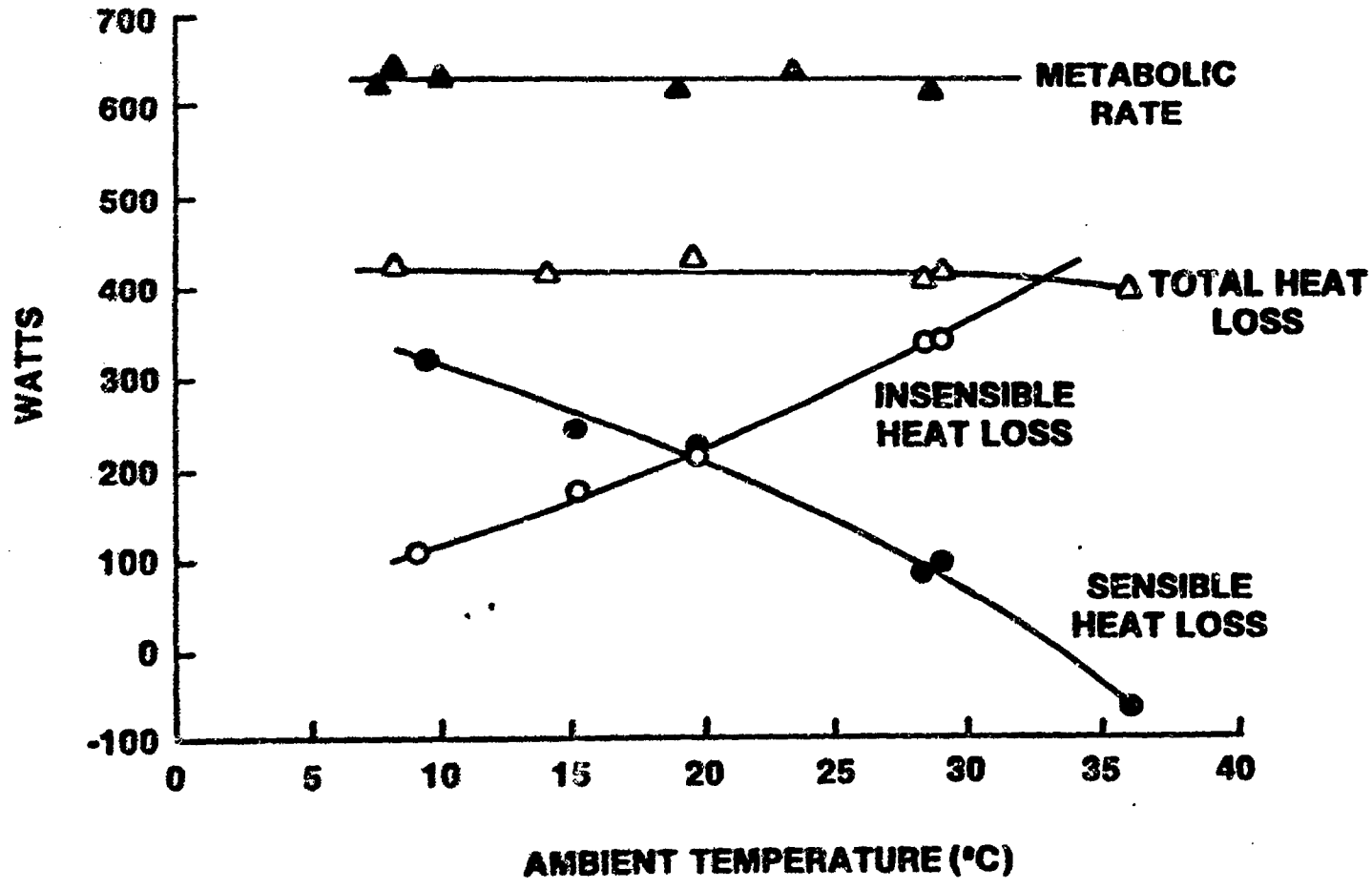

700
600
500
400
300
200
100
0
-100
WATTS
0
5
10
15
20
25
30
35
40
AMBIENT TEMPERATURE (°C)
METABOLIC RATE
TOTAL HEAT LOSS
INSENSIBLE HEAT LOSS
SENSIBLE HEAT LOSS

Fig. 3-5

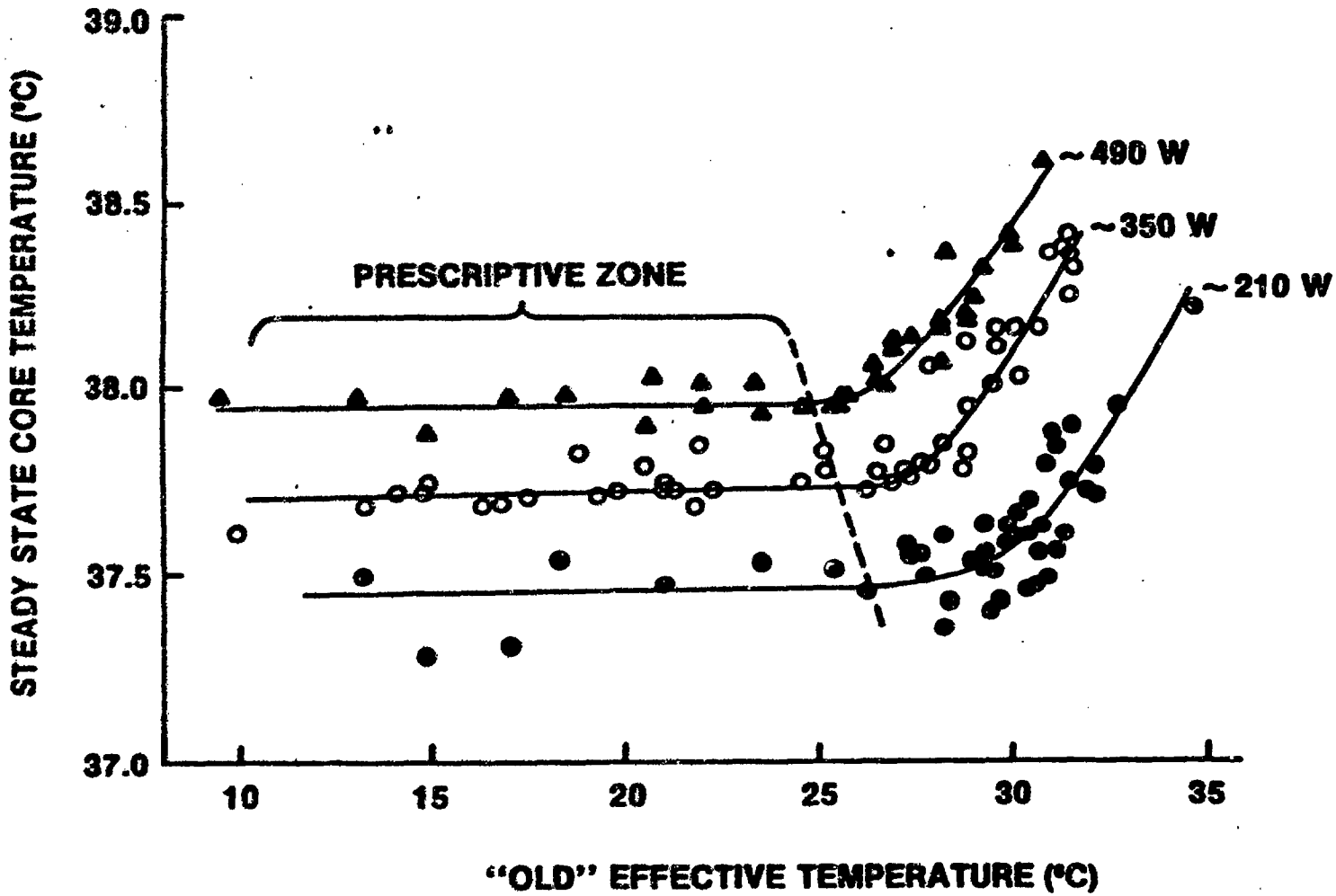

39.0
38.5
38.0
37.5
37.0
STEADY STATE CORE TEMPERATURE (°C)
PRESCRIPTIVE ZONE
~490 W
~350 W
~210 W
10
15
20
25
30
35
"OLD" EFFECTIVE TEMPERATURE (°C)

Fig. 3-6

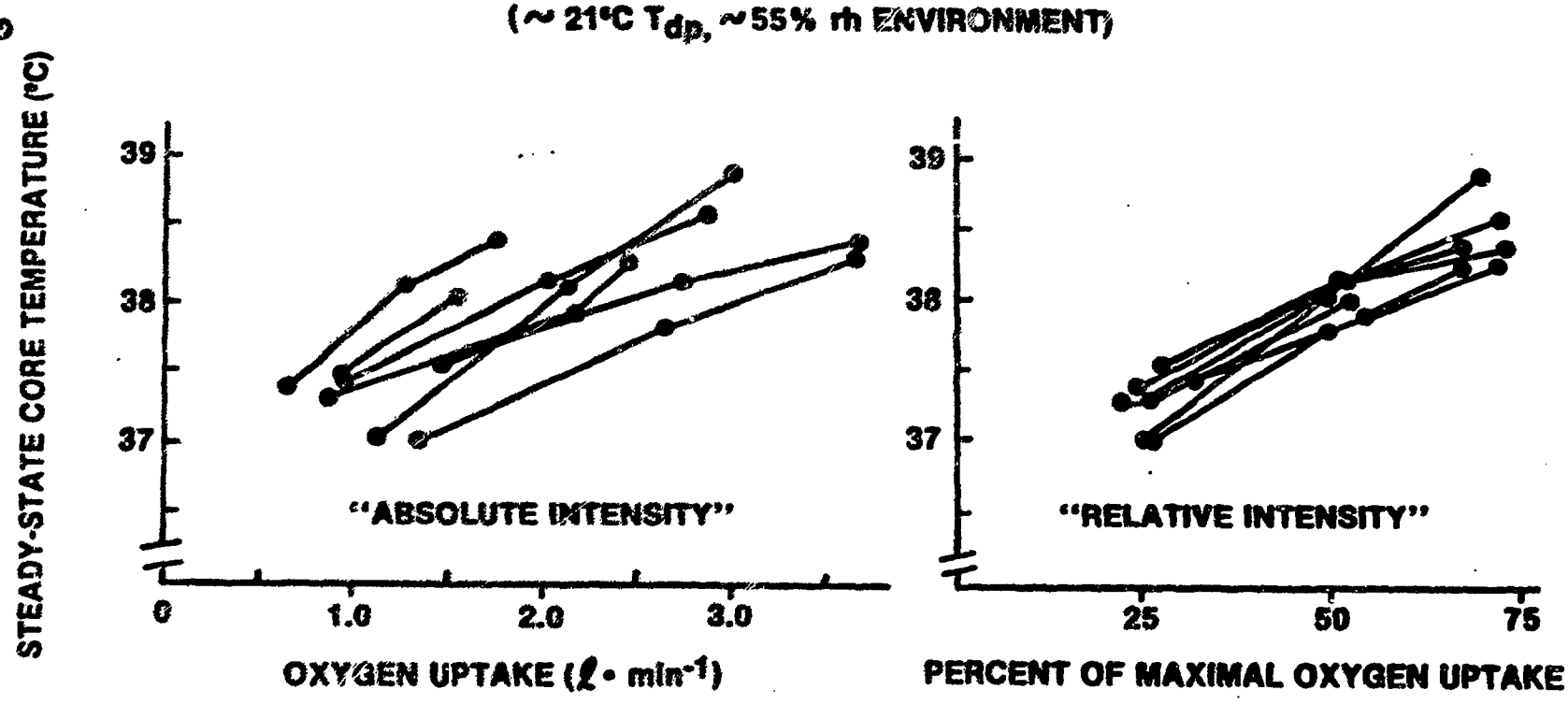

(~ 21°C T_{dp}, ~55% rh ENVIRONMENT)
STEADY-STATE CORE TEMPERATURE (°C)
39
38
37
"ABSOLUTE INTENSITY"
0
1.0
2.0
3.0
OXYGEN UPTAKE (ℓ • min⁻¹)
39
38
37
"RELATIVE INTENSITY"
25
50
75
PERCENT OF MAXIMAL OXYGEN UPTAKE

Fig. 3-7

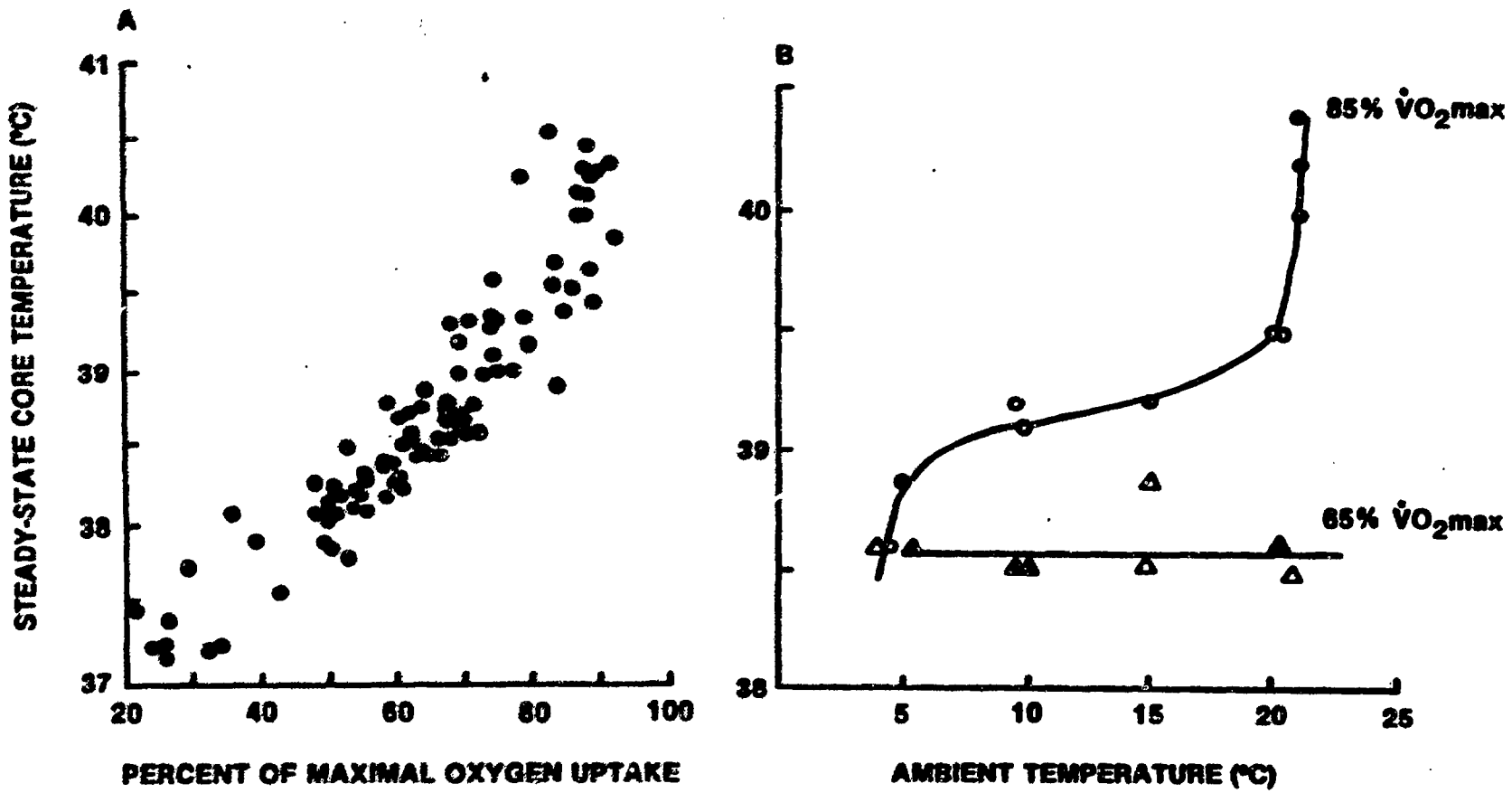

A
B
STEADY-STATE CORE TEMPERATURE (°C)
41
40
39
38
37
20
40
60
80
100
PERCENT OF MAXIMAL OXYGEN UPTAKE
85% $\dot{V}O_2max$
65% $\dot{V}O_2max$
40
39
38
5
10
15
20
25
AMBIENT TEMPERATURE (°C)

Fig. 3-8

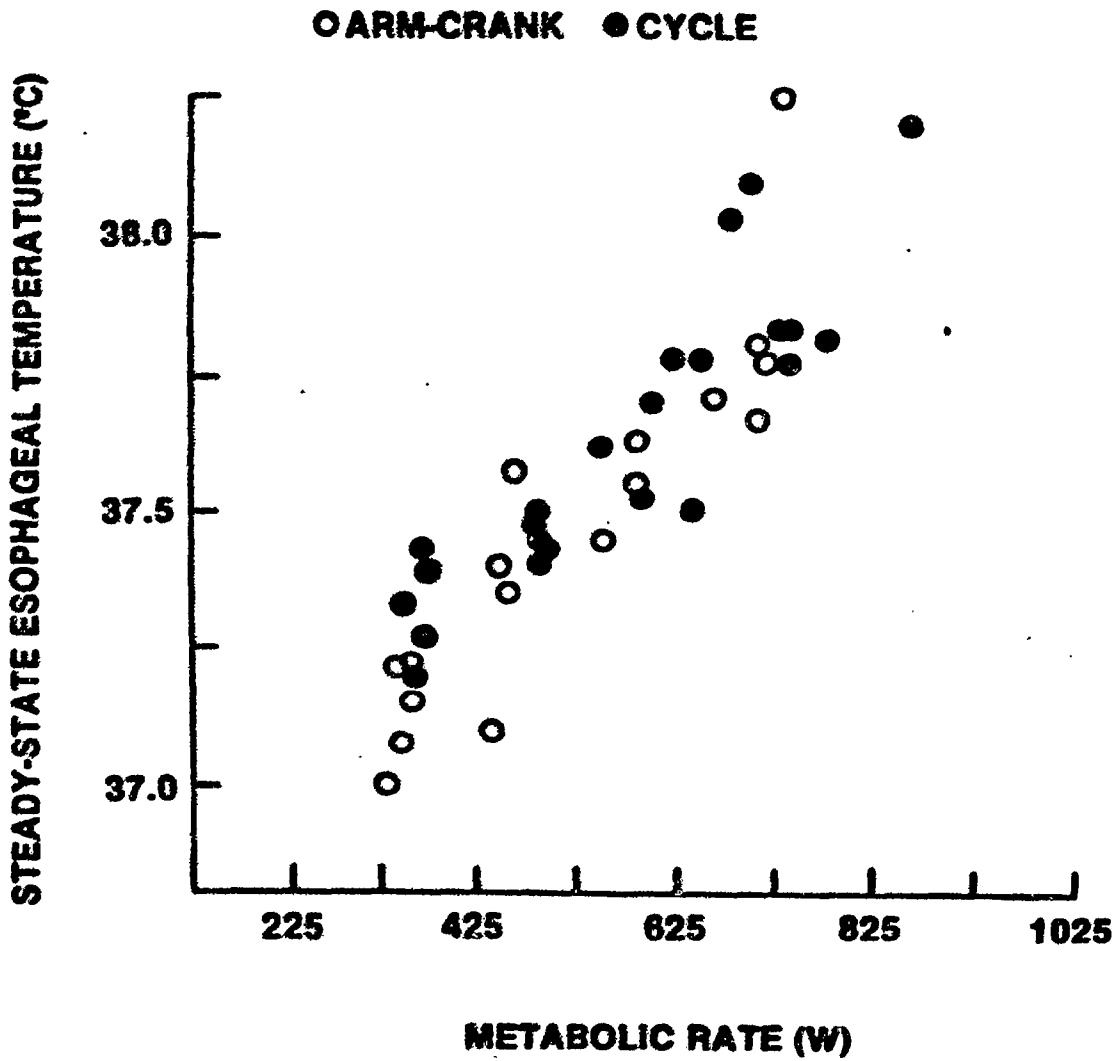

O ARM-CRANK ● CYCLE
STEADY-STATE ESOPHAGEAL TEMPERATURE (°C)
38.0
37.5
37.0
225
425
625
825
1025
METABOLIC RATE (W)

Fig. 3-9

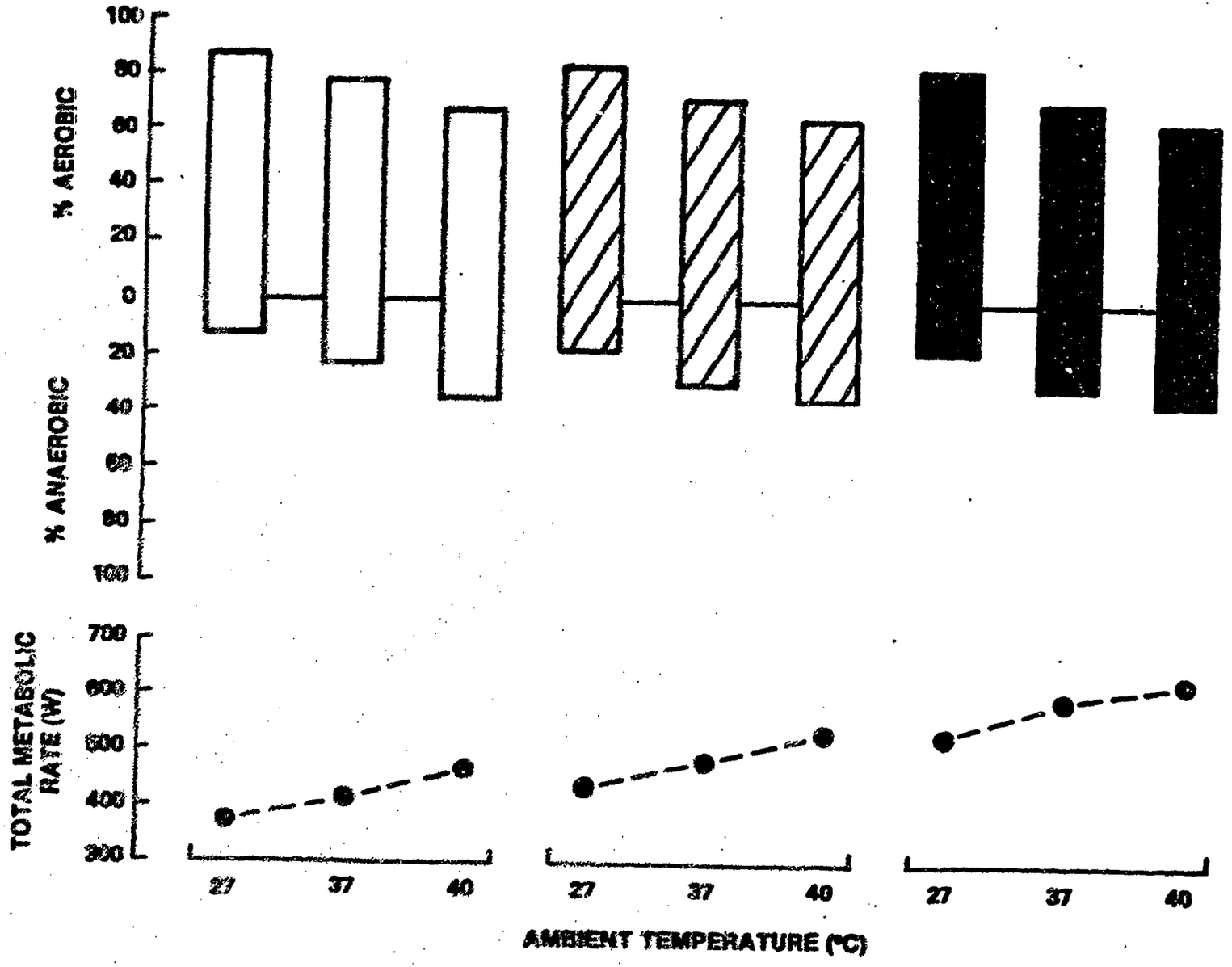

% AEROBIC
% ANAEROBIC
100
80
60
40
20
0
20
40
60
80
100
TOTAL METABOLIC RATE (W)
700
600
500
400
300
27
37
40
27
37
40
27
37
40
AMBIENT TEMPERATURE (°C)

Fig. 3-10

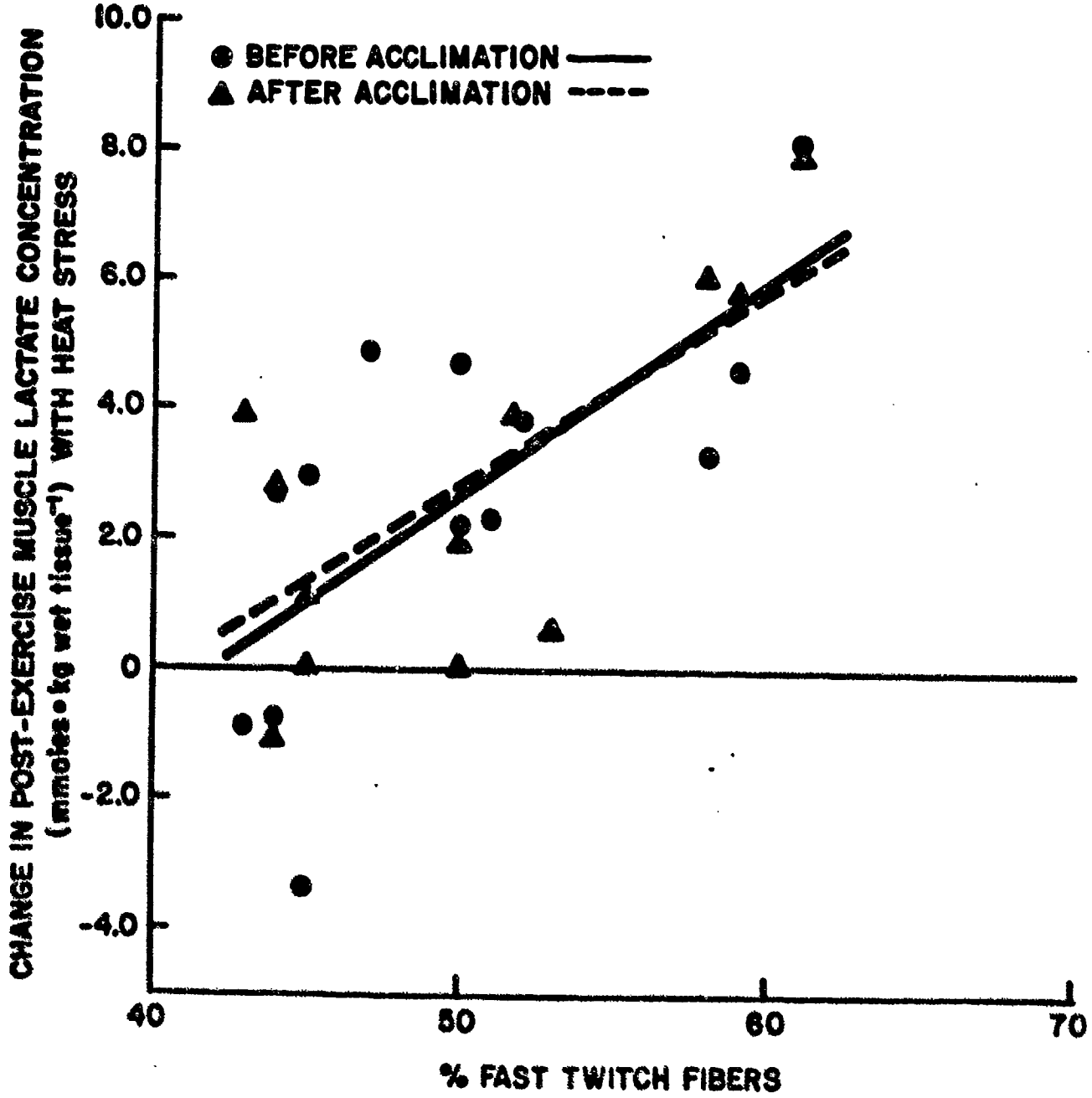

BEFORE ACCLIMATION
AFTER ACCLIMATION
CHANGE IN POST-EXERCISE MUSCLE LACTATE CONCENTRATION (mmoles • kg wet tissue⁻¹) WITH HEAT STRESS
10.0
8.0
6.0
4.0
2.0
0
-2.0
-4.0
40
50
60
70
% FAST TWITCH FIBERS

Fig. 3-11

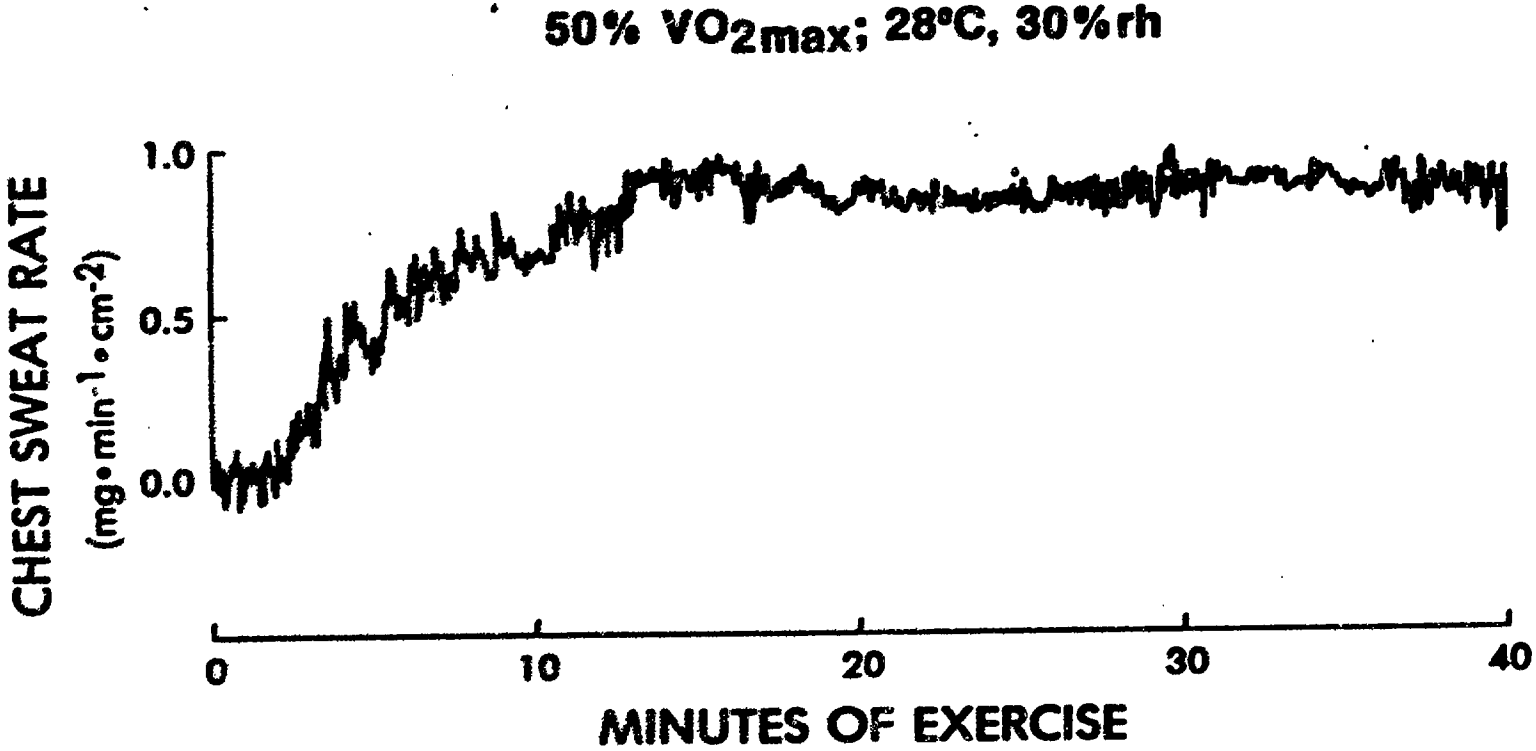

50% VO2max; 28°C, 30%rh
CHEST SWEAT RATE
(mg•min-1•cm-2)
1.0
0.5
0.0
0
10
20
30
40
MINUTES OF EXERCISE

Fig. 3-12

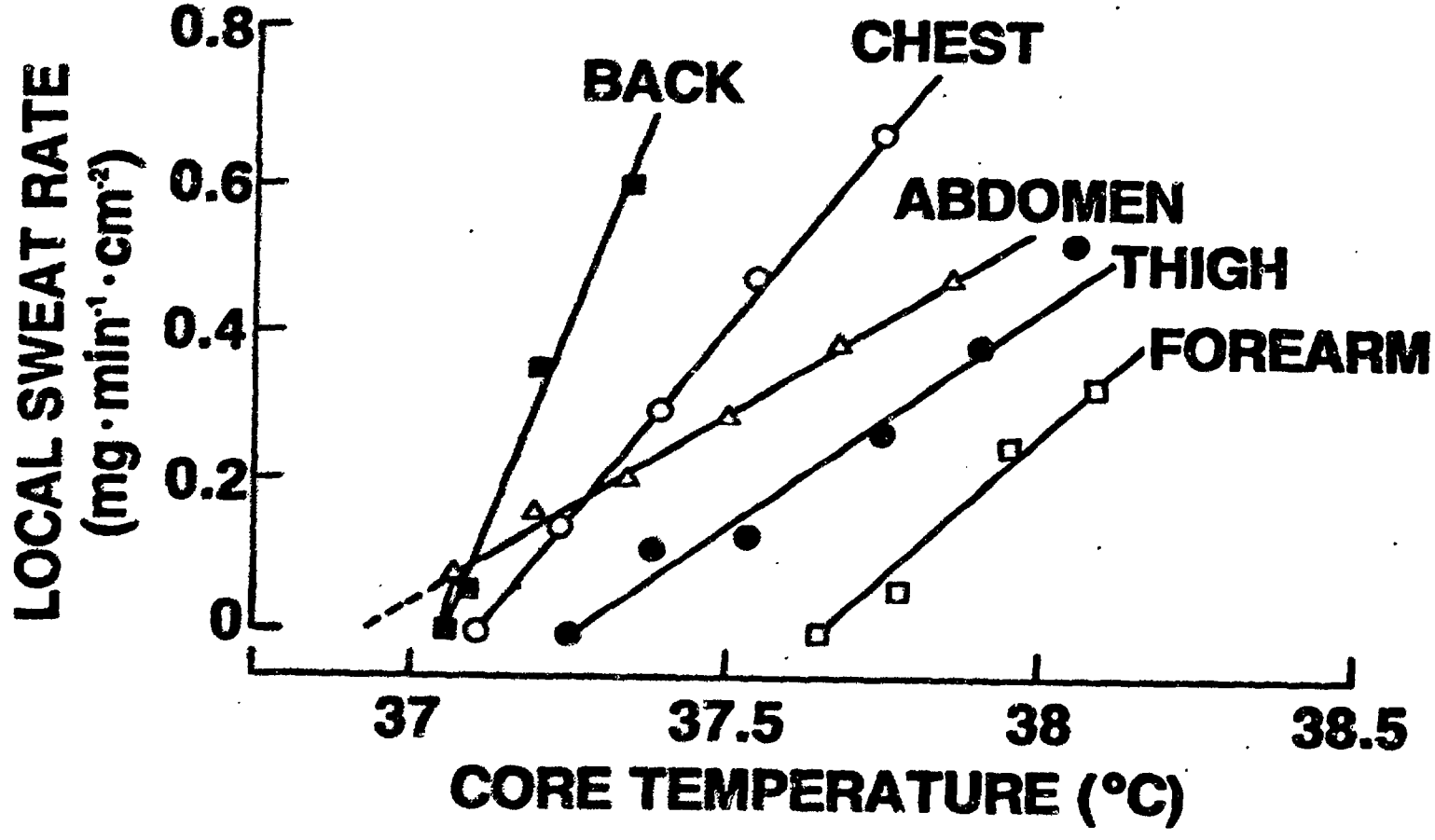

LOCAL SWEAT RATE
(mg·min⁻¹·cm⁻²)
0.8
0.6
0.4
0.2
0
BACK
CHEST
ABDOMEN
THIGH
FOREARM
37
37.5
38
38.5
CORE TEMPERATURE (°C)

Fig. 3-13

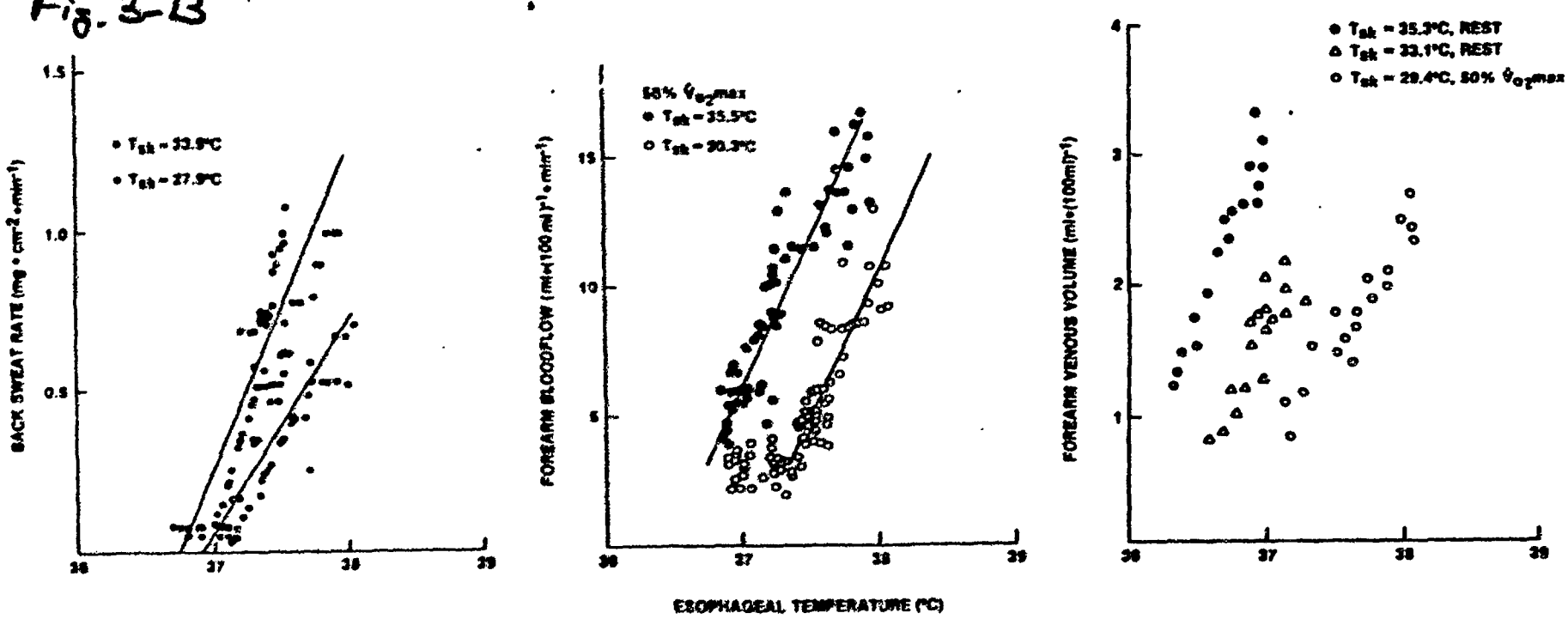

BACK SWEAT RATE (mg • cm⁻² • min⁻¹)
• T_{sk} = 33.9°C
• T_{sk} = 27.9°C
1.5
1.0
0.5
36
37
38
39
FOREARM BLOODFLOW (ml•(100 ml)⁻¹ • min⁻¹)
58% $\dot{V}_{O_2max}$
• T_{sk} = 35.5°C
○ T_{sk} = 30.3°C
15
10
5
ESOPHAGEAL TEMPERATURE (°C)
FOREARM VENOUS VOLUME (ml•(100ml)⁻¹)
• T_{sk} = 35.3°C, REST
△ T_{sk} = 33.1°C, REST
○ T_{sk} = 29.4°C, 50% $\dot{V}_{O_2max}$
4
3
2
1

Fig 3-14

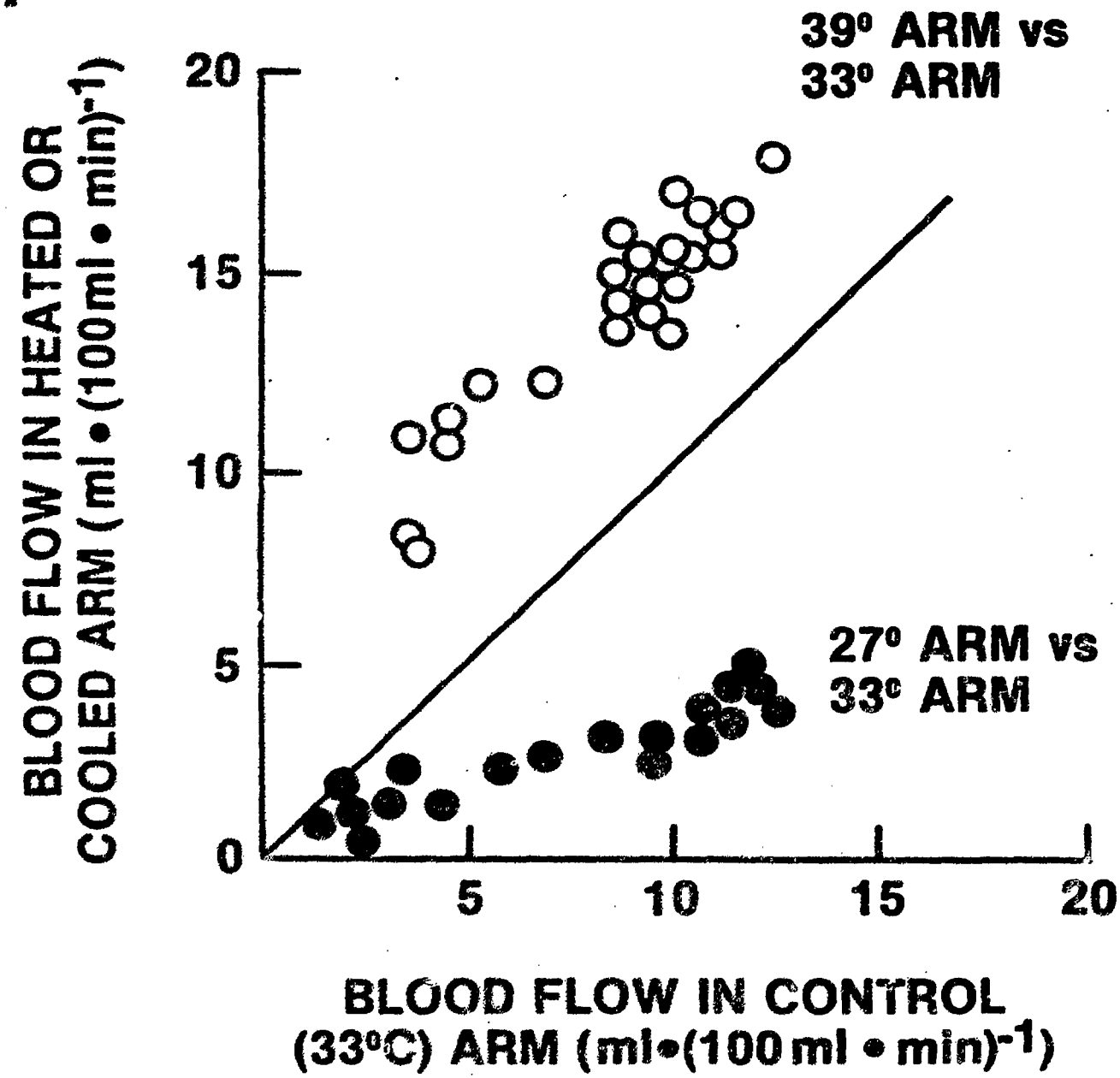

39° ARM vs
33° ARM
27° ARM vs
33° ARM
BLOOD FLOW IN HEATED OR
COOLED ARM (ml • (100ml • min)-1)
BLOOD FLOW IN CONTROL
(33°C) ARM (ml • (100 ml • min)-1)
0
5
10
15
20

Fig. 3-15

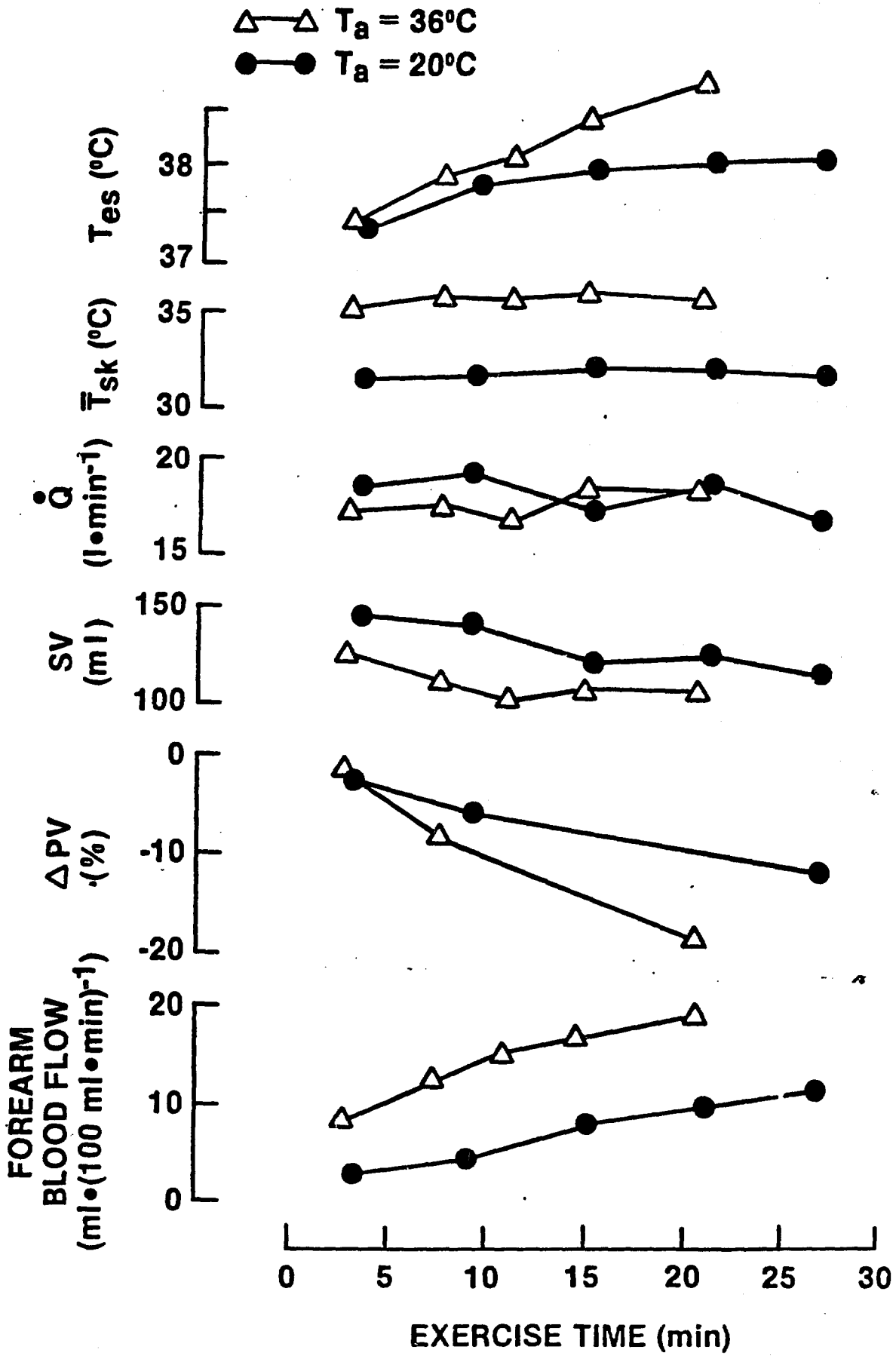

T_a = 36°C
T_a = 20°C
T_{es} (°C)
38
37
$\bar{T}_{sk}$ (°C)
35
30
$\dot{Q}$ (l•min^{-1})
20
15
SV (ml)
150
100
ΔPV (%)
0
-10
-20
FOREARM BLOOD FLOW (ml•(100 ml•min)$^{-1}$)
20
10
0
0
5
10
15
20
25
30
EXERCISE TIME (min)

Fig. 3-16

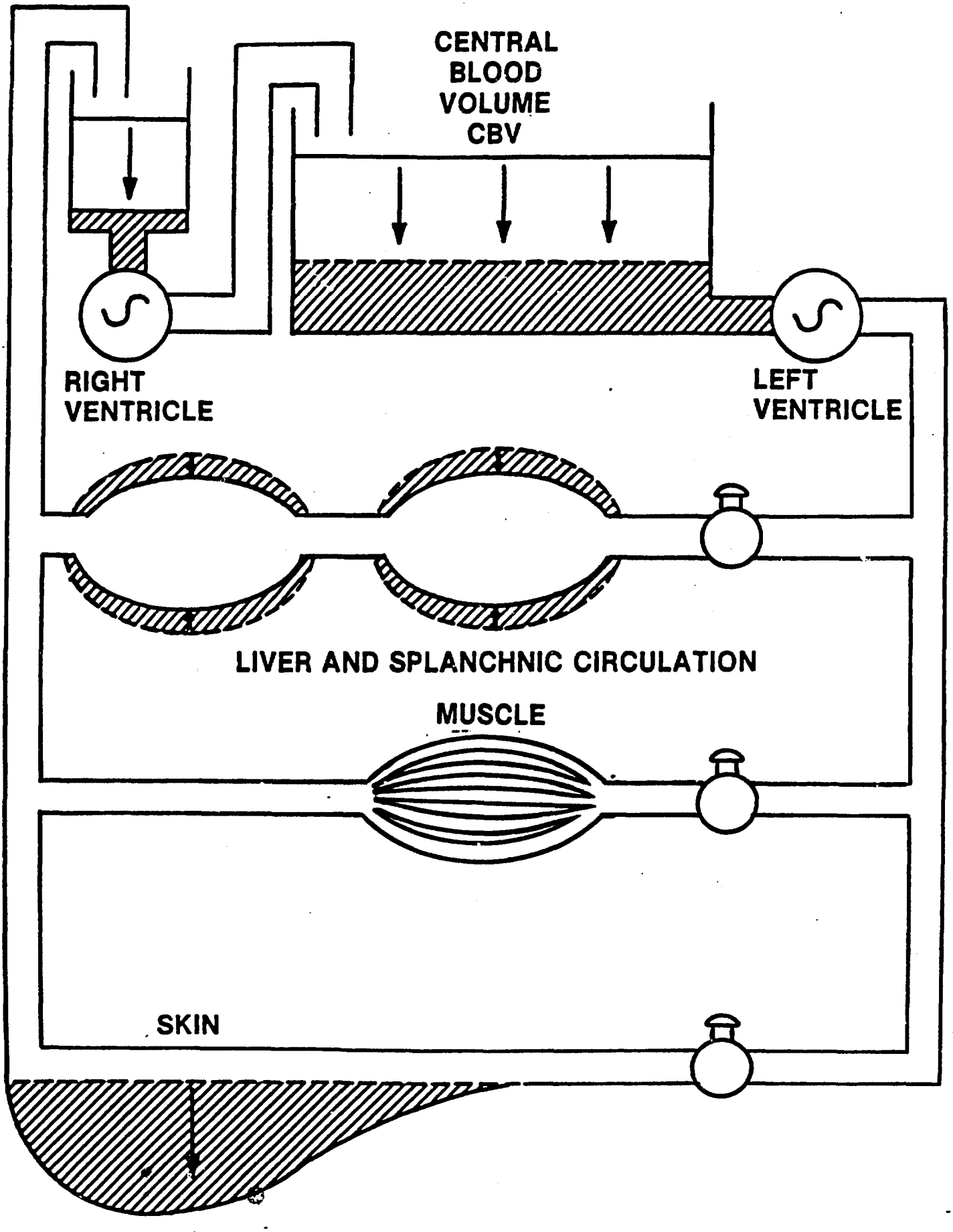
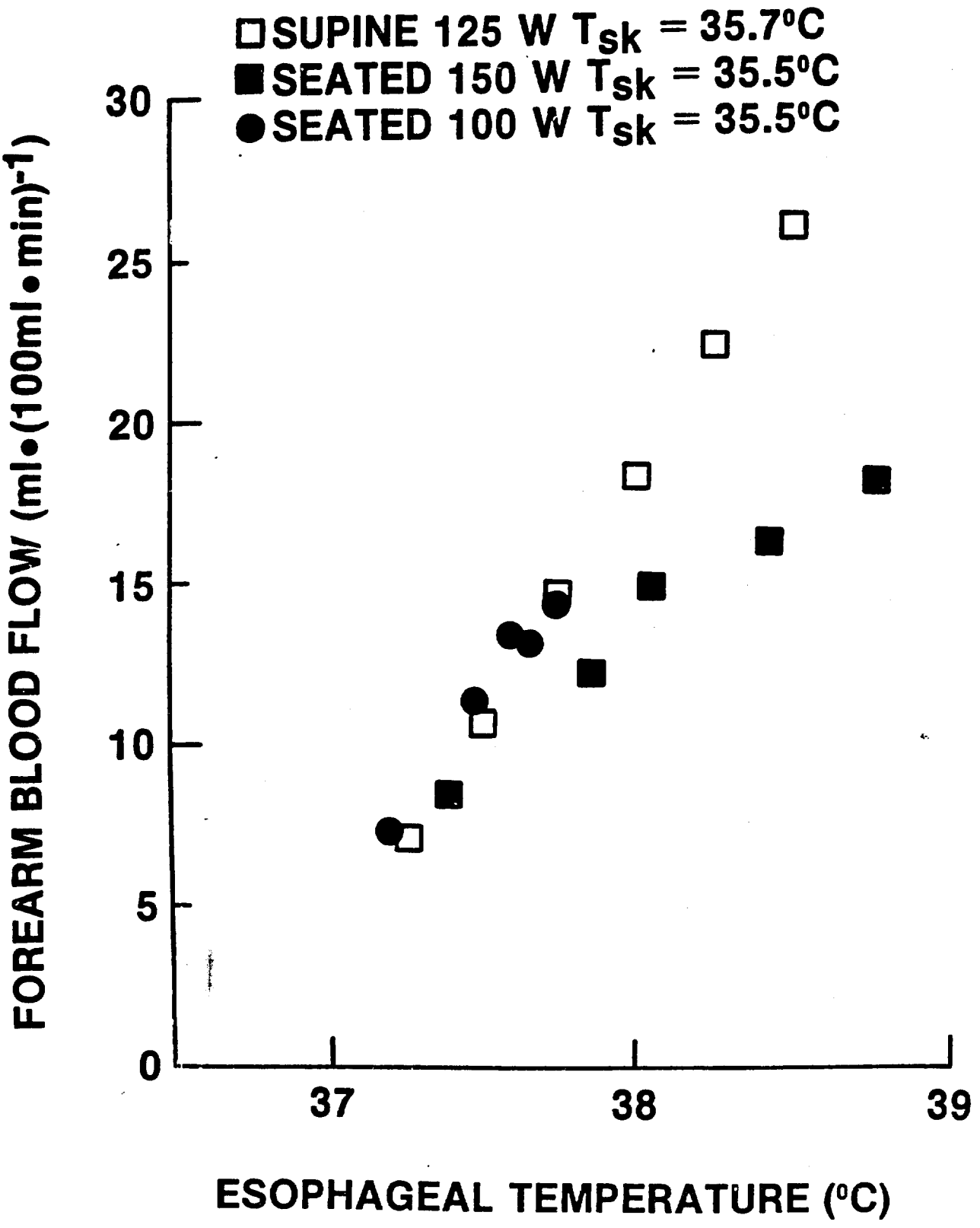

CENTRAL
BLOOD
VOLUME
CBV
RIGHT
VENTRICLE
LEFT
VENTRICLE
LIVER AND SPLANCHNIC CIRCULATION
MUSCLE
SKIN

Fig. 3-17


□ SUPINE 125 W T_{sk} = 35.7°C
■ SEATED 150 W T_{sk} = 35.5°C
● SEATED 100 W T_{sk} = 35.5°C
FOREARM BLOOD FLOW (ml•(100ml•min)$^{-1}$)
30
25
20
15
10
5
0
37
38
39
ESOPHAGEAL TEMPERATURE (°C)